VETERINARY MEDICINE

JUNE 1946

University of Michigan
MAY 22 1946

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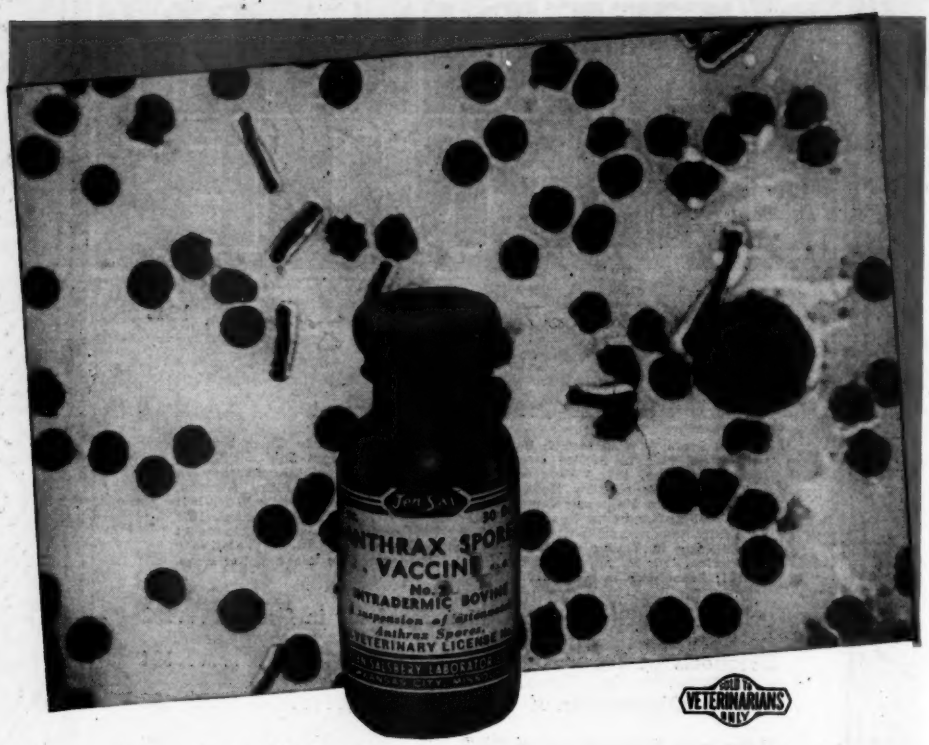
VOL. XLI
No. 6

Entered as second-class matter Nov. 8, 1920, at the post office at Chicago, Ill., under the Act of March 3, 1879.

7812 S. Cranden Ave.
Chicago 49, U.S.A.



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KANSAS CITY, MISSOURI

VETERINARY MEDICINE

Registered in U. S. Patent Office

VOL. XLI JUNE, 1946 No. 6

Published Monthly by the
VETERINARY MAGAZINE CORPORATION
7632 S. CRANDON AVE., CHICAGO 49, ILL., U.S.A.

$4.00 a year; per copy 50 cents; Special Issues $1.00

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Entered as second-class matter Nov. 8, 1920, at the post office at Chicago, Ill., under the Act of March 3, 1879. Entered as second-class matter under the title of American Journal of Veterinary Medicine July 19, 1910, at Chicago, Ill., under the Act of March 3, 1879.

D. M. CAMPBELL, *Editor*
7632 S. Crandon Ave., Chicago 49, Ill.
Telephone South Shore 7160

H. J. MOSTYN, *Eastern Representative*
P. O. Box 121, Sussex, New Jersey
Telephone Sussex 2F18

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That the 1946 beef production in Kansas could be increased by 84 million pounds by treating the 2,800,000 head of cattle in the state with DDT, is claimed by Dr. E. G. Kelly, extension entomologist at Kansas State College.

Soybean oil meal as the only source of supplementary protein in a chick starting ration, produced heavier chicks at eight weeks of age than did peanut oil meal or wheat germ meal, according to scientists of Cornell University.

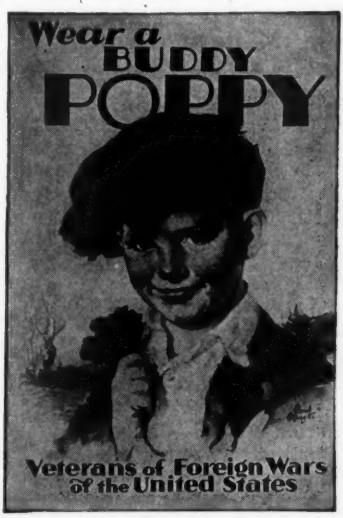
The supply of streptomycin is limited. Requests for it for civilian use should be addressed to Dr. Chester S. Keefer, Evans Memorial Hospital, 65 East Newton, Boston.

Doctor Keefer is chairman of the committee on Chemotherapeutic and Other Agents of the Division of Medical Sciences, National Research Council. When penicillin was new and scarce the allotments for distribution for various uses were made through this committee.

In 1803 stores in which meat was purchased in a survey conducted by the American Meat Institute, from 77% in Newark, N. J., to 94% in Houston, Texas, charged over-ceiling prices. In the New York metropolitan area the average illegal overcharge on 72% of the beef was 40%. On 65% of the pork it was 28%. The A.M.I. concludes as a result of this study that an average of 20 cents of each dollar the housewife spends for meat in 11 large cities is a tax levied by the black market. Only exceptionally was the retailer keeping the illegal overcharge. In most instances he was a victim of the wholesaler or slaughterer.

Buy U. S. Savings Bonds—to have and to hold

The Pennsylvania department of Agriculture inspected more than 1300 slaughter-houses during January and February and closed seven of them for being insanitary. About 40 were in unsatisfactory condition but cleaned up on orders of the state inspectors.



The proceeds of the sale of Buddy Poppies are devoted solely to further the relief extended to veterans and their dependents. Of the various uses to which they are put, none has a more poignant appeal than the V.F.W. National Home for Widows and Orphans at Eaton Rapids, Michigan. A portion of the proceeds of the sale are allotted each year to the maintenance and expansion of this Home. This sale is approved by the President of the United States

Brucella Melitensis in Iowa Swine

It was stated last month *(Vet. Med.*, May, 1946, p. 151) that Brucella organisms of the melitensis strain had been isolated from a number of persons in Iowa who were suffering from undulant fever and swine were suspected of being the source of the infection.

Since the foregoing was reported this suspicion has been confirmed by the finding of *Br. melitensis* in 12 sows belonging to a farmer who was affected with undulant fever. Four of these infected sows were reactors; eight were negative to the agglutination test. Dr. Sam H. McNutt isolated the organisms from the infected swine, Drs. J. A. Barger, C. C. Franks and E. H. Pick cooperated with the state health commissioner and the Director of the State Hygienic Laboratory in the investigation.

Military Deaths in World War II

The Metropolitan Life Insurance Company places the number of combat loss of belligerents in World War II at nearly 10,000,000 of which 5,200,000 were suffered by the Axis and 4,500,000 by the United Nations. The losses of the principal countries involved are estimated to have been: Germany 3,250,000, Japan 1½ million, Italy 150,000, Russia 3,000,000, British Empire 400,000 and United States 325,000.

Military Awards

Legion of Merit

For exceptionally meritorious conduct in the performance of outstanding services during the periods indicated, the Legion of Merit was awarded to the following officers of the Veterinary Corps of the Regular Army:

Col. Seth C. Dildine—Oct., 1941, to Sept., 1945.

Col. Burton A. Seeley—Jan., 1941, to Sept., 1945.

Col. Raymond Randall—Sept., 1939, to Sept., 1945.

Col. George J. Rife—July, 1942, to Sept., 1945.

Col. John L. Owens—Jan., 1942, to March, 1945.

Col. Harold E. Egan—Jan., 1941, to Dec., 1945.

Col. Christian W. Greenlee—Sept., 1940, to Jan., 1946.

Col. Russell McNellis—March, 1942, to May, 1945.

Maj. Don L. Mace—Dec., 1941, to Aug., 1945.

Army Commendation Ribbon

Maj. Terry S. Ozier*—Jan., 1945, to Jan., 1946.

*It is reported that the Congressional Committee on Unamerican Activities will investigate this citation for a veterinary officer not a member of the Regular Army. The report is probably unfounded.

Groth Heads Animal Disease Research Laboratory

April 22, 1946 Dr. A. H. Groth was appointed director of the Regional Animal Disease Research Laboratory at Auburn, Alabama. He succeeds Dr. B. T. Simms who became chief of the Bureau of Animal Industry in November 1945.

Dr. A. H. Groth

Doctor Groth was graduated from the Animal Husbandry Division at Iowa State College in 1921. After he had taught and taken graduate work at Colorado State College and Texas A & M, he returned to Iowa State College and received his degree in veterinary medicine in 1931. He served six years on the Minnesota Livestock Sanitary Board before going to Louisiana State University in 1937. He represented Louisiana State University on the Council of The Regional Animal Disease Research Laboratory at the time of his appointment as its director.

The Unending War

The fight to cripple or destroy medical research and with it medical progress goes on perennially. At present efforts of the "antis" are centered on legislation to prohibit the use of dogs in animal experimentation, but proponents make no secret of their intention to direct the attack against the use of other animals when they have won as to the dog.

Dogs have been selected simply as an entering wedge in the onslaught on all medical research based upon animal experimentation. It is deemed the high regard in which the dog is held by the public eases the task and the extensive exploitation of the use of dogs by the armed forces in the recent war make this an opportune time to push antiscientific measures.

Measures to prohibit the use of dogs in animal experimentation were recently defeated in the Massachusetts and New York legislatures but by margins so narrow as to constitute a warning rather than a triumph for the forces of progress.

Appalling Conditions in India's Dairy Industry

A report to the government of India by Mr. R. A. Pepperall, chief executive officer of the British Milk Marketing Board, reveals appalling conditions in India's dairy industry. The efficiency of the industry is considered by the author to be the lowest in the world. Adulteration by adding water to milk was usual at all stages of distribution from producer to consumer. In Calcutta the added water exceeded 25%. The conditions of milk production in Indian stables are described as "starvation of stock, squalor, filth, ignorance or indifference toward hygienic principles, and revolting personal habits, allied to official apathy." At one Bombay stable housing hundreds of animals, in which conditions might be regarded as normal, the author observed crows feeding from a dead buffalo and then drinking from milk containers. At another stable the buffaloes were allowed to immerse themselves daily in a backwater in which was deposited the urine of 500 animals and in which there were dead animals. Udders were not washed before milking. Official reports showed a bacteria count of 36 million per cubic centimeter for Bombay milk. Although milk production in India is declining, the population is increasing. The price of milk is the highest in the world. The author points out the unparalleled dimensions of the problem of reorganizing an industry consisting of 22 million illiterate and poverty stricken producers with semistarved livestock.—*J. Am. Med. Assn.*, 130:14, p. 963, 1945.

A milk supply in the condition described, non-existent meat inspection, the prevalence of every known disease of animals and sweeping epizoötics of rinderpest, surra, foot-and-mouth disease, piroplasmosis and other animal plagues are a part of the price that India pays for the refusal of the government to develop an adequate veterinary service. There are competent veterinarians in India and several veterinary colleges capable of training many more, but the pay of those in the government service, which includes a majority of all the qualified veterinarians in the country, is so low and the conditions surrounding private practice are so unsatisfactory that the profession is unattractive to students and the veterinary colleges are poorly patronized.

Notwithstanding the All India Veterinary Association has memoralized the governing authorities repeatedly they have persistently refused to employ the services of veterinarians to good advantage or to take the measures necessary to develop a veterinary service adequate in personnel or authority to the needs of its disease ridden livestock industry, to safeguard the general food supply, to assure a dependable food supply or to conserve the work animals of agriculture. The unnecessary loss of animals from disease is no doubt an important factor in the present famine in that country. Periodical famines are a part of the experience of all countries that endeavor to produce their own food without a veterinary service to safeguard the health of their animals.

* * * *

Parasite Control*

The objective of the parasite is to live in amity with its host; obtaining food and shelter without either harming or being harmed. The death, or serious illness, of the host is an equal tragedy for the parasite.

It takes a tremendously long time for an animal to learn the parasite mode of life and become adjusted to survival of itself and host.

In the domesticated animal host-parasite relationship, as in so many other relationships, man has upset nature's balance. By fences and walls he has confined animals where the parasites can infest them readily, and in great numbers.

We know most of the parasites. The life cycles of a majority of them are also known. However, of their distribution, we know comparatively little.

Parasite control measures no doubt antedate civilization. One may observe a manual method in almost any monkey cage at the zoo.

Medical methods for the control of parasites are at least as old as medical history—perhaps much older. A single drug that will eradicate all parasites from a man or an animal was early sought. We are still looking for it. Many weird concoctions have masqueraded in this role. Pumpkin seed is an ancient and crankcase oil a very modern impostor in this field.

Until about 25 years ago medication was held forth as the chief means of parasite control, but we now realize that medication alone can never adequately control more than a few species of parasites; chiefly those that live on or in the skin. Medication will probably always be a necessary part of parasite control, but we are beginning to realize that other control methods may be equal to or superior to the pill, the powder and the liquid parasite killer or crippler.

Medical control possesses many disadvan-

*Notes from an address on "Nutrition in Relation to Parasite Control," by Dr. E. A. Benbrook, Ames, Ia., at the annual Short Course for Graduate Veterinarians, University of Minnesota, October 31 and November 1, 1945.

tages: (1) It is used after damage is done. (2) Most parasiticides are toxic for the host. (3) It is costly in time and money. (4) In adequate concentration, it can be given only periodically. (5) It is too often given without adequate diagnosis. The livestock owner should be convinced there is more to parasite control than buying and administering a drug. There is no greater abuse than giving a toxic parasiticide without a diagnosis—e.g., periodical worming.

Surgery has slight value in parasite control. In a limited field, it is useful in warble and whipworm infestation and in myiasis. Soil disinfection has possibilities. So has manure sterilization by chemicals.

Hygiene and sanitation are not popular as means of parasite control. They are not sufficiently spectacular nor are they in accord with tradition. Nevertheless, separating the well from the sick, the proper disposal of the carcasses of dead animals, circumspection in adding purchased animals to the herd, improvement of buildings, manure removal from buildings and corrals, pasture sterilization where the winters are cold or the summers hot all have value in parasite control.

There is only one worm that completes its life cycle without leaving the host. We have an opportunity to break the chain in all other parasites when they leave the host.

What an animal eats may influence to no small degree its resistance to parasitism. The higher the nutritional level of the animal the greater its resistance to parasites. If the livestock raiser will control infectious diseases and avoid malnutrition in his animals, he will have gone a long way toward solving his parasite problem. Medication should be looked upon as only one means to this end.

* * * *

Avian Pneumoencephalitis

Avian pneumoencephalitis, also reported as a respiratory-nervous disorder (California, 1940) and as Newcastle disease (England, 1927), is an infectious disease involving principally the respiratory organs and the central nervous system of chickens, turkeys, ducks, geese, pheasants and quail. The disease is indigenous in many parts of the world but had not been diagnosed in the United States until recent outbreaks occurred in California (1940) and New Jersey (1945). As yet avian pneumoencephalitis has not been recognized in Illinois. However, veterinarians and poultry raisers are urged to be on the lookout for this disease.

Cause.—Avian pneumoencephalitis is caused by a specific filterable virus. In the early stages of the disease, the virus is present in the lungs, spleen, tracheal exudate, brain, blood, intestinal contents and feces of affected birds. Birds of all ages are susceptible.

Symptoms.—Generally, severe respiratory as well as nervous symptoms are manifested. In chicks, outbreaks of pneumoencephalitis have begun as a respiratory disturbance, characterized by rales, coughing and gasping, which is indistinguishable from respiratory symptoms observed in infectious bronchitis. Involvement of the central nervous system begins a few days after the onset of the respiratory trouble. Inability to coördinate voluntary movements, partial or complete paralysis of one or both legs, incoördination of the neck muscles, and tremors of the head and jerky movement, not all of which are exhibited by the same bird, are typical of the nervous symptoms of this disease. Cases with involvement of the central nervous system continue to appear in an affected flock even after respiratory symptoms subside. In older chickens the prominent symptoms are difficult respiration, coughing, gasping, loss of appetite and nervous symptoms similar to those observed in chicks. Mortality may be slight or severe. In laying flocks, egg production is markedly reduced and may cease entirely.

Spread.—The disease is rapidly spread from infected birds to healthy chickens by contact. Chickens that apparently have recovered may be virus-carriers. Feces from infected birds carried on the footwear and clothing of the attendant may further disseminate the virus on the premises. The initial source of the infection often is obscure.

Diagnosis.—The disease does not produce any constant and characteristic lesions recognizable on post-mortem examination. A cloudiness of the air sac membranes is the most common lesion. The presence of combined respiratory and nervous symptoms in a flock is a valuable lead in recognizing the disease. Laboratory procedures are necessary for demonstration of the virus by transmission tests or in chicken embryo culture. Serological tests also are proving valuable in establishing a diagnosis.

Control.—No medicinal treatment of value is known. Isolation of affected birds and proper disposal of the dead are essential measures in controlling an outbreak. Birds surviving an attack should be sold. Houses and equipment should be cleaned thoroughly and disinfected. Encouraging results have been secured in recent experiments (California) in immunizing chickens against this disease. It seems reasonable to expect the development of an effective avian pneumoencephalitis vaccine.—*An. Path. Exch., Univ. of Ill.*

Baby Pig Mortality*

The loss of pigs between farrowing and weaning is, as is well known, very large. Year in and year out it probably averages 25% of the pigs that are born apparently healthy and with a reasonably good chance to survive. The most critical period in the pig's life seems to be the first six to ten days. Pigs that survive approximately one week after they are born acquire a stamina and a tolerance that enables them to endure influences that often prove mortal to younger pigs.

Many swine raisers fail to realize the extent to which starvation figures in loss of young pigs. Even vigorous newborn pigs will often die within 36 to 48 hours if deprived of food soon after birth. Partial starvation, combined with chilling, will kill many of them as promptly during this period and in turn, severe chilling alone will kill them unfailingly.

Prevention of starvation in pigs should begin with the sow. Of mature sows only those that are known to be good milk producers should be bred. When gilts are selected for breeding only those with regularly spaced teats, and no blind teats, should be used for breeding. Further, the sow, whether gilt or mature, should receive an ample balanced ration right up to farrowing. The sow should be rather fat when she farrows. Sows differ in milk production as much as do cows and they suffer from agalactia too. An occasional sow apparently produces too much milk for her litter and as a consequence, her pigs may acquire enteric troubles from overeating. Her ration should be reduced. A far greater number supply an insufficient quantity of milk for their newborn litters and this is frequently the starting point for a high mortality.

If the sow has agalactia during the first few days after farrowing, the pigs usually become comatose within 36 to 48 hours and die. If she suffers from retained placenta or a uterine discharge, as is common in herds in which there is Brucella infection, she usually supplies insufficient milk and the pigs may starve. In these latter cases, the farmer often believes that the milk is poisonous or "acid" or infective and is responsible for the death of the pigs, but as yet investigation has failed to reveal anything harmful in the milk. Generally, there just isn't enough of it.

The pigs from such sows can be raised on cow's milk if the owner is willing to go to the trouble to hand-feed them, and it is trouble to hand-feed pigs because they must be fed regularly throughout the 24 hours, at least during the first few days. If these pigs, already about on a balance between life and starvation, are not fed during the night, many of them will be dead in the morning.

Newly-born pigs when starved or receiving a markedly insufficient diet develop hypoglycemia. In the course of a few hours severe hypoglycemia does irreparable damage to the nervous system and death is inevitable. However, if they are given repeated injections of glucose (25% solution) within an hour or two of the onset of coma and are warmed to normal body temperature, many of them can be saved.

Dr. C. C. Morrill of our staff has found that pigs held experimentally at a temperature of 15° C. (59° F.) and fasted, developed intense hypoglycemia in about 24 hours. If, however, they were kept at 31° C. (88° F.) and fasted, they did not develop an intense hypoglycemia until the lapse of approximately 80 hours. These observations clearly demonstrate the extreme importance of providing warm quarters for newborn pigs, especially if the pigs are not getting enough milk.

*Notes from an address, by Dr. Jesse Sampson, at 40th Annual Meeting of the Mississippi Valley Veterinary Medical Association, Galesburg, Ill., Nov. 15-16, 1945.

The Importance of Mortality in Young Pigs

Few farmers appreciate what pig losses cost them annually. It has been reliably estimated that not more than 56% of the pigs farrowed alive ever reach market. The average hog raiser's sows farrow 100 pigs to wean 65 to send 56 to market. He pastures, houses, feeds, and cares for two sows to do the job of which one is capable. He wastes an enormous amount of labor and feed each year because of these pig losses.

The average pig costs at least 100 lb. of feed at birth. Even worse than the death losses at or soon after birth are those that occur later. According to some authorities, each pig that lives to be ten weeks old and then dies causes a loss of from 250 to 275 lbs. of feed. Each one that dies at eighteen weeks of age wastes from 350 to 375 lbs. of feed, while each one that dies at six months of age may waste 600 lbs. of feed. This mortality of 44% between farrowing and market wastes 14% of our hog feed annually. Since feed cost amounts to 85% of the total cost of producing pork, this means that these mortality losses make up about 12% of the total cost of producing our pork. To reduce these mortalities by half would reduce the cost of pork production by 6%. This is of utmost importance because at least one-half of these mortalities can be avoided. Put another way, this means a waste of from 2½ tons to 3 tons of feed annually for the average hog producer who maintains ten sows.—*Purina Nutr. News Bul.*

Achievements of the Federal Veterinary Service

In a speech in the House of Representatives, March 8th, Congressman Gillie of Indiana paid a high tribute to the Bureau of Animal Industry and the Meat Inspection Service of the U.S.D.A. Excerpts from Doctor Gillie's speech follow:

The Bureau of Animal Industry and the Meat Inspection Service are constantly at work inspecting animals and food and conducting research and experimentation to insure a healthy and disease-free livestock industry and a steady stream of healthful food to our tables.

* * * * * * * * *

It is impossible in a short time to outline in detail the work being done by the Bureau in meat and food inspection. Bureau veterinarians work in packing houses, in butter factories, in dairies, in warehouses. They even inspect livestock while it is on the farm and before it has been slaughtered for marketing, and carry on from there until the food is sold to us over the counters of our stores.

Perhaps the most spectacular accomplishment of the Bureau has been in the field of livestock improvement. Largely due to Bureau efforts during the more than 60 years of its existence, our livestock industry is the largest and healthiest in the world. Tribute should be paid to the fearless leadership of men such as Salmon, Melvin, and Mohler, who headed the Bureau during its most turbulent years. Several national campaigns against animal diseases were conducted under their leadership considerably in advance of public opinion in many areas. Lawsuits, violence, and even bloodshed marked the progressive application of veterinary science. In more recent years, I am glad to say, public opinion has been highly receptive to these campaigns.

* * * * * * * * *

In the Bureau's scientific research there has been more devotion to achievement than fanfare of results. Entanglements of old traditions and smoke screens of skepticism have sometimes impeded application of the new knowledge, but in spite of obstacles, progress has been continuous. Take, for instance, the painstaking research on worm parasites of hogs and the development of the now familiar swine sanitation system.

* * * * * * * * *

Let us examine another illustration. Back in 1917 when the national compaign against bovine tuberculosis began, about 50,000 cattle a year failed to pass Federal meat inspection. Instead of qualifying for human food, those animals went for fertilizer, grease, or tankage. By contrast, the number failing to pass inspection in recent years has been less than 2,500—in other words an annual saving of about 47,500 cattle, equivalent to some 24,000,000 pounds of dressed beef.

Thus we see how the Bureau's foresight a quarter of a century ago, plus good organization and courageous effort, is now paying food dividends in a hungry world.

* * * * * * * * *

Bovine tuberculosis is just one of several disease enemies now practically conquered. Cattle tick fever is another, sheep scab is another, and pullorum disease of poultry is another.

* * * * * * * * *

You can see, therefore, how closely tied up are human health and livestock health.

These are only a few of the many jobs Bureau veterinarians are engaged in. Progress against other important maladies is a matter of public record. Congress, in providing funds for such activities, is performing a real service to agriculture and to the Nation.

As I have said on a previous occasion to my colleagues in Congress, we are now literally holding in trust for the rest of the world the best blood lines now extant for the reestablishment of disease-free herds and flocks in all those war-stricken countries.

* * * * * * * * *

There has been need for a great deal of research—to find improved systems of breeding, to develop more useful types of animals, to test new feeds, to reduce losses, to save labor, and to enhance quality.

* * * * * * * * *

The appropriation requested for the work of this Bureau during the coming fiscal year is not large. Viewed as a dollar-and-cents investment in our livestock and food industry it is good business. It will pay big dividends to farmers and consumers in the form of improved herds and farm products of the highest quality.

* * * *

Michigan Mastitis Control Program

Mastitis is any inflammation of the udder, irrespective of the cause or severity. Acute mastitis may be caused by non-infectious or infectious factors. Chronic mastitis also may be caused by non-infectious or infectious factors.

Mastitis Program

The mastitis control program is available to every dairyman of Michigan. There are no rules, regulations or agreements to sign. It is the responsibility of each dairyman to see that the testing is properly carried out and certain essential sanitary measures are adopted in handling his herd, if the program is to accomplish anything of value in his dairy cows.

Frequency of Testing Cows for Mastitis

The number of times that the herd should be tested will depend on the individual dairy herd. A suggested plan is to test at monthly intervals for three months, every other month for about eight months and at about three to six-month intervals thereafter. The tendency is to allow an interval of time too great between tests. This is unfortunate for not all cows of the herd are lactating at any one time and only milk from lactating cows can be tested with any degree of accuracy, and if infection gains entrance to the herd, it may spread and do much damage before its presence is detected, if tests are made only at long intervals.

Prevention, Control and Treatment of Mastitis

(Consult your local veterinarian early)

A. *Good, sanitary milking procedures.*

1. Arrange and/or milk cows in following order: healthy, suspected and infected.

2. Discard the foremilk into a strip cup, exclude from supply all abnormal milk.

3. Wipe the udder of each cow with warm (120° F.) disinfectant one minute before milking.

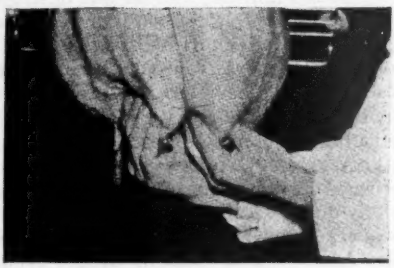
4. Exclude infected persons from milking, handling the milk or milk utensils.

5. Dip teat cups of milking machine and hands of the milker in disinfectant between each two cows.

6. Milk each cow regularly, rapidly, and completely for udder health.

B. *Good sanitary herd management procedures.*

1. Have proper stalls for cows.
2. Treat the udder and teat injuries promptly.
3. Clean stalls upon removal of infected cows.
4. Keep barn and barnyard clean and dry.
5. Raise heifers as disease-free replacements.
6. Remove accessory teats from heifer calves.
7. Procure replacements preferably in this order: (a) home-raised heifers, (b) purchase



Make a physical examination of the udder of each cow

of bred heifers and (c) purchase of milking cows. Test before purchase.

C. *Early and accurate diagnosis depends upon:*

1. Physical examination of the udder of each cow.
2. Bacteriological test to determine that infection is present or absent.

D. *Elimination by slaughter or complete segregation of infected cows with badly damaged udders.*

1. Such cows usually do not respond to udder infusion treatment.
2. This eliminates or limits the source of infection.

E. *Early and proper treatment.*

1. A high percentage of cows, without marked change in the udder, recover when given udder infusion treatment.
2. Udder infusions are of value only where infection is the cause of the mastitis.

. Control measures must accompany treatment or cases that recover will become reinfected.—*C. S. Bryan.*

Breed Better Hogs

Faster-growing hogs of superior conformation and greater vitality are resulting from experiments of the Regional Swine Breeding Laboratory, Ames, Iowa, conducted by the U. S. Department of Agriculture and 13 State agricultural experiment stations. The research involves carefully planned systems of matings in which inbreeding has a prominent part.

Swine raisers, using their farms as proving grounds, are cooperating with the scientists by testing boars produced by the various systems of breeding. More than 500 boars from the various projects have been released to farmers, for practical tests of breeding excellence. Five breeds and three lines from crossbred foundations are represented in the research, and the degree of inbreeding in most of the lines ranges between 25 and 50% and in some instances higher.

Experiment station data, together with reports from farmers, indicate that mating boars from good inbred lines to non-inbred sows results in about 100 pounds more weight per litter, when the pigs are six months old, than that of litters sired by non-inbred boars of the same breeding as the sows. Crosses involving three inbred lines, all within the same breed, are a promising method of growing still more pork per mating. Preliminary trials of this method have resulted in the survival of 6% more pigs to the age of eight weeks and about 300 pounds greater litter weight, at market size and condition, than were obtained from good purebred stock not inbred. The experiments indicate that the greatest increase in vigor and growth rate comes from crossing inbred lines developed from unrelated foundation stocks.

Decline in vigor and fertility, commonly associated with inbreeding, is counteracted largely by improved methods of selection. Any decline which does occur is overcome by the crossing of animals representing different inbred lines within the same breed. The progeny are known as "crossline" animals. Research is being continued to purify further the lines of breeding and to arrange for more widespread use of the stock by the swine industry.

* * * *

In an UNRRA report Dr. M. M. Kaplan, Chief of the veterinary section in Greece, states that the soil of Greece is heavily contaminated with anthrax and tetanus organisms, that about 80% of the livers and lungs of sheep and goats, seen in the abattoirs, contain hydatid cysts indicating that approximately 95% of the farm dogs harbor *Echinococcus granulosus* and that this tapeworm is frequently transmitted to human beings in that country.

UNRRA Livestock Importation Problems in Greece

by MARTIN M. KAPLAN, V.M.D., M.P.H.*
Chief, Livestock and Veterinary Section,
UNRRA Greece Mission
Athens, Greece

THE importation of livestock into Greece is of great importance in the restoration of Greek economy shattered by five years of war and occupation. Ruined harbors, bombed docks, disrupted communications, feed shortages, few veterinarians and ever-threatening enzootic diseases greatly enhance the normal complexities of receiving large numbers of animals in a relatively short period. Operational difficulties have been overcome to a large extent in Greece, but the question of disease control does not lend itself to a solution by means of trucks and cement.

The spotlight falls immediately upon the diseases indigenous to a country supplying livestock. Thus far there has been imported into Greece mules bought from the British Army in Italy, mules and donkeys from Cyprus, and mules, mares, heifers and bulls from the United States. During the current year (1946), however, several other countries will supply various types of livestock which will call for more strict supervision than ever, if Greece is to be spared the added burden of new veterinary communicable diseases, or increasing reservoirs of infection in already established diseases. A graphic example of the latter danger is the present situation in respect to foot-and-mouth disease.

Since June, 1945, much effort has been expended in keeping foot-and-mouth disease localized in Thrace. The outbreak started on the Turkish border in eastern Thrace and has gradually spread westward to the Nestos river, which, as a natural barrier, has helped to prevent the disease from spreading into Macedonia and possibly to all of the mainland of Greece. While our eyes were focused on the danger spot in Thrace, a boat carrying infected cattle from Turkey landed on our back doorsteps in Piraeus in January, 1946. UNRRA had advised against all cattle importations from Turkey and other countries of the Middle East, until reliable veterinary information can be obtained from those countries. However, a private dealer imported animals adorned with aphthous fever vesicles which escaped the supervision of a negligent veterinary inspector. The consequences need no elaboration for one who is even theoretically acquainted with the communicability of the foot-and-mouth disease virus.

Mules brought from the British Army in Italy offered very little trouble. They were selected animals to begin with, mallein tested, and in excellent physical condition. Most of them had been procured originally from Cyprus and other countries in the Middle East. They had been in Italy since early 1944 which minimized the danger of carrying the African horse sickness virus against which they had been vaccinated while held in Palestine. In the quarantine stations in Greece, mange proved to be a bit troublesome but much more serious was the finding of epizootic lymphangitis. One laboratory proved case of this disease was found and the animal was destroyed. It can only be hoped that too many cases did not escape inspection in the various ports.

Mules and donkeys from Cyprus have been satisfactory from the veterinary point of view.

Importation of mules and mares from the United States presents the problems of equine infectious anemia, equine encephalomyelitis, and virus abortion, none of which have been definitely diagnosed in Greece. It is reasonable to believe, however, that equine infectious anemia does, in fact, exist in Greece because of its known presence in the neighboring countries of Bulgaria and Yugoslavia. No assurance against importing chronic carriers of the infectious anemia virus can be given because of the diagnostic difficulties. Concerning the introduction of the United States strains of equine encephalomyelitis, most of the evidence points against the persistence of the equine encephalomyelitis virus in solipeds *in a manner favoring communicability* after the first few days of illness. Nothing is known about the status of virus abortion of horses and asses in Greece. Its importation from the United States is possible, but because of diagnostic difficulties little can be done to eliminate infected or carrier animals.

Brucellosis is enzootic in Greece, but the prevalence is not known. Whenever possible, cattle imported from the United States are inoculated with Strain 19 brucellosis vaccine.

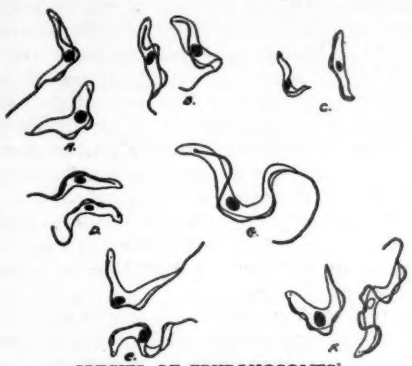
Importation of horses from Central and South America poses the question of the trypanosomiases (*Trypanosoma equinum, T. hippicum,* and *T. equiperdum*). The complement-

*Associate Professor of Preventive Medicine and Public Health, Middlesex University School of Veterinary Medicine, Waltham, Mass.

fixation test provides the only practical, precautionary measure, although the desirable three tests made three weeks apart are difficult of execution for large numbers of export animals. It is probable that only one test may be all that it will be practicable to make. It should be noted that dourine is enzootic in Central Greece (Thessaly) and Macedonia (around Salonika).

The presence of foot-and-mouth disease in some countries of South America demands care on the importation of cattle from those sources. Greece has enough trouble on this score without starting new flare-ups of possibly different strains of the virus. It is not likely that many cattle will be procured from these countries. If any are procured it has been requested that they be from areas free of foot-and-mouth disease for the past 1½ years.

South Africa will probably not be tapped as a source of livestock for Greece because of the danger of rinderpest, surra, nagana, and Afri-



SPECIES OF TRYPANOSOMES[1]

A. *T. brucei* B. *T. vivax* C. *T. congolense*
D. *T. equinum* G. *T. theileri*
E. *T. equiperdum* F. *T. evansi*

can horse sickness. An exception may be made for solipeds from the Union of South Africa which is known to have a competent veterinary service. Further, a dependable vaccine against African horse sickness is produced by the veterinary laboratory at Onderstepoort. During the war the British Army shipped over 24,000 mules vaccinated against African horse sickness from South Africa to India, and the disease has not appeared in the latter country. (The presence of the insect vector, *Culicoides,* has not been established in Greece.) The vaccine contains an attenuated neurotropic strain of the virus prepared from brains of inoculated mice. A negative mallein test and complement-fixation test for trypanosomiasis will be required of horses and mules from South Africa.

Syria and Egypt may be used as sources of breeding stallions. African horse sickness has been reported in both these countries and UNRRA is attempting to arrange protective

Edmonds

African horse sickness has been reported in Syria and Egypt

inoculation of any stallion procured. These animals will be subjected also to the mallein and complement-fixation tests.

The greatest difficulties in shipping livestock are encountered with pregnant heifers and pregnant mares. Mules appear to withstand even long sea voyages quite well, provided they are given a plentiful supply of fresh, clean water and not too much concentrated feed. Prophylactic inoculations of hemorrhagic septicemia antiserum appear to be quite beneficial in mitigating shipping diseases in contrast to the poor results obtained when bacterin is used. The greatest mortality in mares is caused by pneumonia secondary to strangles. The mortality rate from this cause alone has ranged between 1% and 5% in different shipments. Abortions in pregnant mares and heifers are not uncommon during the voyage, especially when rough weather is encountered.

Upon landing in Greece all animals are held in quarantine for two weeks. Necessary treatments and protective inoculations are given during this period. Horses, mules and burros usually receive a second dose of tetanus toxoid, the first having been given prior to shipment, and they are also inoculated with anthrax intradermal spore vaccine. Cattle are inoculated against anthrax and blackleg.

Summary

Some problems concerning the procurement, shipment, and reception of animals by UNRRA for Greece are presented. In spite of the difficulties, the net result has been, and undoubtedly will continue to be, a great boon to the deserving Greek farmers who made unstinting sacrifices for the cause of the United Nations.

Diagnosis of Tuberculosis*

By H. C. KUTZ**
Harrisburg, Pennsylvania

DIAGNOSIS of tuberculosis is based on three main factors: (1) the physical examination, (2) the technic of injecting the tuberculin, and (3) the interpretation of the reaction.

The physical examination is the first step in eradicating tuberculosis from any herd and should not be made in a matter-of-fact manner. Each animal should be examined, paying particular attention to the superficial lymph glands, abnormal type of respiration, general emaciation and any bloating due to enlarged mediastinal lymph glands. The physical examination is of paramount importance especially in herds where a large percentage of the cattle react to the tuberculin test as, frequently in such herds, there may be an animal that is so extensively affected with tuberculosis that the test will not reveal a reaction.

Cattle showing any evidence of tuberculosis on physical examination should be condemned even though they do not react. The veterinarian must be exacting in conducting tests. Any other policy subjects him to criticism.

It is well to keep in mind that, due to the lessened prevalence of tuberculosis, cattle are more susceptible to the disease than formerly and if it be introduced into the herd it may spread rapidly.

The second factor is the correct technic in the injection of the tuberculin. Since 1920, the intradermal test has been the official test. It has proved equally as reliable as the subcutaneous test. It possesses the advantages of permitting the testing of large numbers of cattle in a short period. Were it not for the intradermal test, the cattle of Pennsylvania and all the other states would not all be tested today. It required 22 years to test all the cattle in Pennsylvania. It would have required at least 100 years to test them by the subcutaneous method.

Other advantages of the intradermal test are: It permits testing under all conditions, ages and disposition of the cattle. It pleases the herd owner because it does not require confining the cattle as long as for the old subcutaneous method.

A disadvantage of the intradermal test is its apparent simplicity, which conduces to carelessness on the part of the veterinarian, in that sometimes sufficient time and patience is not taken to make the injection properly.

Before any testing is conducted, the veterinarian should give attention to equipment. Clean overalls and rubbers should be put on before entering the barn, especially in herds that are enlisted in the brucellosis eradication project; the footwear should be immersed in an antiseptic bath. Frequently herd owners complain about the possibility of veterinarians carrying infection from one farm to another.

The syringe used for making the injection should be of the regular tuberculin hypodermic type equipped with a sharp needle of 25-gauge, ¼ or 5/16th of an inch long. A shorter needle does not permit the depositing of the tuberculin into the dermal layer of the caudal fold. Some speak of the tuberculin injecting as a stabbing operation. This is not correct. Such practices may account for some of the difficulties encountered in eradicating the disease from some herds.

Before the injection is made, the veterinarian should have the animal restrained. A proper injection cannot be made if the animal is moving around. The syringe and needle should be cleaned with alcohol and cotton before each injection and filled with tuberculin that is clear and not outdated; cloudy tuberculin is an indication of contamination. Needless to say, the site of injection must likewise be cleaned with cotton and alcohol.

In Pennsylvania we inject on the left caudal fold only; the reason being that in case the veterinarian making the injection is unable to return to the farm to interpret the reaction to the test, another veterinarian may do so. The seat of injection on the caudal fold is about three to four inches posterior to the anus. The needle should be inserted horizontally into the dermis. Care should be taken that the tuberculin is not deposited superficially just beneath the epidermis nor deposited subcutaneously. It is a good practice to examine the fold after injection to be certain that the tuberculin has been properly injected. If deposited in the loose tissues the injection is of no diagnostic value.

In cows we inject also on the lower third of the line of demarcation between the skin and mucus membrane of the vulva. In other words we use the high and low method of injection routinely. In bulls the lower edge of the anus can likewise be injected. Two minims is the proper dose of tuberculin; larger doses break down the tissues and cause false shotlike reactions which may be confusing on observation at the 72nd hour. Indifference and careless technic, such as failure to clean and dis-

*Presented at the Conference of Veterinarians, University of Pennsylvania, Philadelphia, January 9, 1946.

**Chief of the Tuberculosis Division, Pennsylvania Bureau of Animal Industry and President of the Pennsylvania State Veterinary Medical Association.

infect the needle before each injection, reflects unfavorably upon the veterinarian and also upon the profession.

A third factor in the test is the interpretation of the results of the injected tuberculin. Observations are made at approximately the 72nd hour on both the modified accredited area plan and the individual accredited herd plan. At this time some clinical cases may show pronounced systemic reactions. In our area testing injections are made on Monday, Tuesday and Wednesday, and read Thursday, Friday and Saturday. If a herd discloses heavy infection a later reading is made at about the 120th hour to catch any delayed reaction, which sometimes occurs in heavily infected herds.

Generalized tuberculosis

A reactor is indicated by a circumscribed or diffused swelling, hot, painful and discolored, or small infiltrations or nodules. In herds showing extensive reactions all animals showing any abnormal swelling irrespective of its size should be condemned. If this policy is not followed infection may remain in the herd and reactors be disclosed on future tests. Do not have suspects in heavily infected herds. In addition, the physical examination must be painstakingly made in such herds and the history of the herd must be weighed carefully. Has it been assembled recently? When was it last tested? Has the herd disclosed reactors in any previous test? Every angle should be gone over thoroughly. The dams or nurse cows of reacting calves must also be examined carefully, paying particular attention to the udder for tuberculous mastitis. In such cases if a calf shows pronounced reaction the veterinarians would be justified in condemning the dam or nurse cow even though she did not react to the test. If any chickens have access to the cattle barn they should also be considered as it is well known, tuberculous chickens may sensitize the cattle and cause reactions. Also, the health of the hogs should be considered, as garbage-fed hogs are a source of danger. In addition, the health status of the persons who work around the barn, especially those in institutions such as asylums or hospitals must be taken into account as tuberculous persons may cause cattle to react. If there be any doubt in the veterinarian's mind, it is better to condemn an animal than take a chance that might result in the entire herd reacting later. On our area work we have no suspects, and our records show that this policy is the best in the long run.

The size of the reaction swelling is no indication of the degree or extent of infection. Animals extensively affected with tuberculosis may disclose only slight or small reactions and those showing swellings as large as a lemon may show only small lesions on post-mortem examination.

Cases have come to our attention where the owner was so anxious to have his cattle react that he injected an irritant such as turpentine or creolin or used pliers to pinch the caudal fold and cause swelling. However, pseudo-reactors would be so unusual in type and the odor of the injected material might be so evident that the veterinarian should be able to detect it. If such cases should develop, the entire herd should be quarantined for a retest under direct, Bureau supervision and classed as a tampered herd.

Always examine the upper and lower sites of injections for the reason that in certain instances an animal may react on the lower injection and not on the upper.

The bovine tuberculosis eradication program has reduced the bovine infection to one-half of 1% or less in all the states. But we cannot assume that bovine tuberculosis is a thing of the past. We must not conduct tests under a false sense of security.

When we receive post-mortem reports from federal and state veterinarians on brucellosis reactors or butcher cattle indicating tuberculosis, we immediately check the herd of origin and retest the herd either by the owner's veterinarian or one of the Bureau veterinarians. We also advise the veterinarian who makes the test, of the post-mortem findings so that he has a history of the herd before testing, which serves as warning to be on his guard when interpreting the tests.

In one instance 35 reactors to the brucellosis test, when slaughtered, revealed lesions of tuberculosis. Of the 35, six carcasses were tanked. Retests of the herds from which these reactors originated, disclosed 23 reactors of which 16 showed lesions.

In another case 13 butcher cows when slaughtered revealed lesions of tuberculosis with eight carcasses tanked. Retesting of the herds from which these butcher cows originated revealed 16 reactors with one tanked.

Clinical Observation of "1080" Poisoning

By E. J. FRICK and F. W. BOEBEL
Manhattan, Kans.

"1080" is the latest in rodenticides. As a matter of information, "1080" is so called because it was the thousand-eightieth in a long series of materials tested for their toxicity for rats. Chemically the poison is sodium fluoroacetate. It is highly effective as a rat killer but it is also very toxic to other animals. The chief characteristic of this compound is that it is extremely soluble in water. It may be used in very low dilutions and still maintain its effectiveness.

According to E. R. Kalmbach of the Wildlife Research Laboratory, Fish and Wildlife Service, "the new ratbane seems to be the deadliest substance ever tried out for this purpose. In carefully controlled tests, it has been able to kill the common Norway rat in concentrations as low as 5mg per kilogram of bodyweight. It is reasonably certain that the discovery of '1080' assures this nation of a highly effective, economic poison which can not be denied this country through any future interruption of world trade."

If and when this product is released for commerial trade, it will become another problem for the veterinarian. It was with this in mind that the following work was done.

Two horses were used. Before starting the work, both animals were weighed and the dosage was based upon the weight. The amount of chemical given was in proportion to that amount necessary to kill a wild Norway rat. One animal received the substance *per os;* administration to the other was intravenous.

Case One

This animal weighed 1100 lbs. and was given 2.5gm of "1080" in 100cc of water *per os* by means of the stomach tube.

The first symptoms were noticed about one and one-half hours later. The heart rate was increased and the beats were less distinct than before. The animal showed restlessness, considerable pawing and extreme hyperesthesia of nostrils. About 20 minutes later, sweating was added to the symptoms. The mucous membranes were very pale. The pulse rate greatly increased. One hour later, there was still marked depression with a slight urticaria, sweating and some shivering.

Seven hours after administering the chemical, the body temperature had dropped below normal. The mucous membranes showed marked congestion. They were a bright, brick red in color. There was grinding of the teeth and marked depression. The ears and nose were getting cold. There was a constant urination. The "lubb" part of the heart beat was like a beat of the bass drum, very strong. Whereas the "dubb" part was very low and suppressed.

Approximately 15 minutes after the above symptoms were noted, the animal went down. It was dead within three hours after it had gone down. During the three hours that it was down, the heart beat was so irregular one could not obtain a count. The respiration had become very rapid. The mucous membranes remained markedly congested. The animal struggled little if any. There were practically no violent movements at any time.

On post-mortem examination there were no visible lesions that could be said to be characteristic of this poisoning, except one. That was the color of the blood. It was black, thick and tar-like.

Case Two

This animal was a thoroughbred weighing 850 pounds. It was given 0.9gm of "1080" in 30cc of water intravenously. One hour later another dose of the same amount was given in a similar manner.

The first symptoms were noticed one hour following the second dose. They were extreme nervousness, pawing, hyperesthesia of the nostrils, and the mucous membranes were beginning to show congestion.

Two hours later, the animal was sweating excessively. The perspiration was coming off the skin in very large drops. Again there was marked depression and some trembling. About one and one-half hours later, the sweating subsided a little but the trembling appeared to have increased.

Five hours after the second dose was administered, the animal was dead. The mucous membranes showed a marked congestion. Again there was no evidence of struggling or violence. There was uncontrolled urination.

On post-mortem examination there were no visible characteristic lesions except the black, thick, tar-like blood.

Carcass Poisoning

Some muscle meat, liver, and heart tissue was taken from the cadaver of case 2 and fed to three dogs.

Feed had been withheld from these dogs for 56 hours. Each dog when fed had all it wanted to eat of the particular tissue assigned to it. Throughout the entire time of the experiment the dogs had water available to drink.

One dog ate 2.6 pounds of muscle tissue. The second dog ate 0.8 pound of liver and the third dog, 2.8 pounds of heart. Six hours after feeding the meat from the poisoned animal, the dogs were observed and all appeared to be normal.

Eighteen hours after feeding, the dog that had eaten the heart tissue was dead. The other two still appeared normal six days later.

On post-mortem examination, the dog that died showed no characteristic lesions except the black, thick, tarry appearance of the blood.

From this experiment one may say that "1080" poisoning:

1. Seems to exert its action primarily on the heart.

2. It is a slow acting poison as far as solipeds are concerned.

3. It is a very toxic chemical as indicated by the small amount that proved lethal—the lethal dose was 5mg per kilogram bodyweight.

4. Heart muscle from poisoned animals transmits the poison to other animals that may consume it. Apparently the poison has an affinity for cardiac tissue.

5. That there was practically no struggling or violence shown by any of the animals used in the test.

* * * *

Uses and Limitations of DDT*

DDT is a synthetic product made from benzene, chlorene, ethyl alcohol and catalized by sulfuric acid (2, 2-bis (p-chlorophenyl)-1, 1, 1-trichloroethane). The forms from which insecticides are made are DDT purified and technical DDT. The purified form is used in aerosols. Sprays and dusts for household pests, agricultural pests and pests of man and other warm blooded animals are made from technical DDT.

DDT has received more publicity than any other insecticide ever introduced. Most of this stems from its wide and exceedingly effective use by the Army. However the peculiar conditions under which the Army employed it are rare in civilian life. Its place in our everyday economy is by no means established. Scientists are reluctant to make any general recommendations as to its use. The prospects are that when rotenone is again plentiful DDT will fade out of the picture except for its use against flies and mosquitoes in buildings where advantage can be had from its residual effect. In fact it is already being elbowed from the center of the stage by benzene hexachloride.

*Notes from an address by Prof. F. E. Guyton on DDT in Relation to Man and Animals and Some of Their Insect Pests at the 21st Annual Conference for Veterinarians, School of Veterinary Medicine, Alabama Polytechnic Institute, Auburn, Feb. 26 and 27, 1946.

Forms of DDT on Market

DDT appears on the market in many forms and under various trade names. For instance, Geigy Company calls their agricultural form "gesarol" and their household and animal form "neocid." Du Pont calls their agricultural and animal form "deenate" and their industrial form with the same formula "deenol." Niagara Company's trade name is "nitox," with DDT not even mentioned; and so it goes for a dozen or more companies, each having its own trade name. These various forms are:

A. *Solutions.*—Usually 5% DDT in oil.

1. Household sprays—for knockdown of flies, mosquitoes, etc.; not recommended.

2. Aerosol bomb sprays such as used in the late war; recommended.

3. Thermal fogs—applied by special fog machines for treating entire herds of cattle at once. Very effective against the horn fly.

4. Residual sprays—where DDT is deposited on surface and insects contact it there. This type has been very satisfactory in barns, kennels, chicken houses, garages, fabrics, insect control, etc. The sprays of aqueous mixtures are very effective for four weeks or longer.

5. Soil poisons for termites—effective for two years so far.

B. *Emulsions*—These are made by using emulsifiable oil solutions of DDT in water. Residual type materials used as sprays or dips for flies, lice, and ticks, and in barns, etc.

C. *Suspensions.*—These are prepared by stirring a wettable or dispersible powder containing DDT in water. May be used in place of solutions and emulsions for residual spraying in most cases. Its objectionable feature is a whitish residue on sprayed surface. A hound dog that was infested with fleas, lice, ticks, and flies was sprayed with this material. Everything was killed but the dog.

D. *Dusts.*—The dusts consist of DDT mixed with a suitable diluent such as talc or pyrophyllite or sulfur, *but never lime.* Normally for household and animals they are used as a 10% dust. They can, however, be purchased in concentrated forms—25-50% DDT and diluted to 10%. They have been effective against many of the pests of the household and of animals.

Toxicity to Animals and Man

It is easy to conclude, from various experiments performed with cows, sheep, goats, rats, and dogs, that DDT is poisonous to warm-blooded animals when taken orally in sufficient amounts. But there is a need for much more experimentation in this field. It has been established that dogs can be killed by eating 100mg per kilogram of body weight, per day six days per week for seven weeks; and that 10mg per kilogram body weight had no dis-

cernible effects on dogs when fed over a period of 50 weeks.

Orr and Mott of the federal Bureau of Entomology, after having fed cows, horses, and sheep up to a maximum of 200mg per kilogram of body weight concluded, ". . . the results of the present tests do not, however, give reason to expect any bad effects on domestic animals from the proper use of DDT as an insecticide when applied as a dust or in water suspension." (U.S.D.A. Bu. of Ent. E673) Goats fed 1.25 and 0.68 grams of DDT per pound body weight transmitted sufficient DDT to their milk to kill white rats in 29 and 31 hours after administration. More DDT was found in the cream than in skim milk. Sprayed goats did not absorb enough DDT to affect suckling kids. (*Science*, Vol. 102, No. 2669)

The U. S. Public Health Service has indicated that 10% DDT dusts and 1% DDT in deodorized kerosene, if properly used, should offer no health hazards to human beings. Most authorities agree that DDT in oil is dangerous to man and animals when the oil is allowed to contact the skin.

DDT has been found toxic to fish when used in concentrations greater than one part in 10 million.

DDT Against Insect Pests

Fleas.—Five or 10% dust used on dogs in small amounts has given complete kills of fleas. It has been suggested that from ½ to ⅓ teaspoonful of the 10% DDT be dusted along the back of the dog. Certainly it should not be placed on suckling bitches or on young puppies. However, their bedding may be dusted, provided the DDT be covered with new bedding. DDT should not be used on cats. A 10% dust is preferable for treatment of fleas in buildings and lawns. In household treatment, dust under the house, behind the baseboards, and under the rugs. If the material is not removed for several days, one treatment should be enough. If fleas are on lawns, use 10% DDT wherever fleas are found. A 5% dust kills fleas in 24 hours; a 10% kills in four hours.

Ticks.—For ticks on dogs, treat lightly with 10% DDT dusts. If ticks infest a dwelling, dust under and behind baseboard and door facings with 10% DDT. Use 10% dust around the outside of house and on the porches. The 5% DDT kerosene spray is effective against ticks.

Flies.—

1. Hornflies on cattle: Use 2.5% DDT (water suspension) at the rate of one quart per adult animal. (Use 2 pounds of 50% water-dispersible powder to five gallons of water, or 4 pounds of 25% to make the above 2.5% concentration.) This should give a high control of the horn fly for 10-15 days on first application and 2-3 weeks or longer following a second application. (U.S.D.A. Bu. of Ent. E-675)

2. House fly: Spray the inside of the chicken houses, barns, kennels, garages or other outbuildings with water-dispersible DDT of the above percentage at the rate of one gallon per 300 square feet (U.S.D.A. Bu. of Ent. E675). Most of the flies will leave the building before dying.

Inside the household, 5% kerosene solution is desirable since it will not leave a noticeable residue. Screens may be sprayed with these materials, but mopping the screen with a sponge will save material. Remember, oil solutions should not contact skin nor be inhaled. *Never lose sight of the fact that proper sanitation is the most effective and desirable method of controlling the house fly.* One ton of stable manure will breed one million flies in 10 days, but chicken manure is really productive. DDT is not very effective against stable flies.

Lice: For lice use a 10% powder on head lice, crab lice, and cattle lice; a 5% powder on chicken lice.

Bed Bugs: On beds, use of 5% oil emulsion at the rate of three liquid ounces of spray per double bed, or two ounces of 10% dust. Remove sheets and spray mattresses. There is no reason to have bed bugs any more unless one really likes them.

Cattle Grub: DDT is relatively ineffective against warbles.

Horse Bots: DDT is relatively ineffective against bots.

Clothes Moths: For fabrics use DDT in a highly volatile oil; it is efficacious and long lasting, even through two or three launderings or cleanings.

Heel Flies, Screwworm Flies, Mites and Chiggers: DDT is relatively ineffective against these pests.

Cautions

1. Always treat DDT as a poison.
2. Where DDT is to be applied extensively, it is well to use a respirator.
3. DDT in oil can be absorbed through the skin. Wash with soap and water immediately if DDT in oil contacts the skin.
4. *Never use DDT in oil on animals.*

Some Publications You Might Like to Read

1. Use of DDT in control of flies on cattle and around farm buildings. H. H. Stage, Bu. Ent. E-673.
2. Summary of DDT Experiments on Insects That Affect Man and Animals. W. E. Dove, Bu. Ent. E-673. These two bulletins may be obtained from U.S.D.A. Bureau of Entomology and Plant Quarantine, Washington 25, D. C.
3. DDT and Its Uses. La. Ext. Serv., Jan. 14, 1946. Supplement No. 1, Baton Rouge, La.

Histamine Theory and Treatment of Laminitis*

By JEAN CHAVANCE
Saint-Florentin, France

1. Theory

READING the remarkable work of J. Tinel on the sympathetic nervous system inspired our histaminic conception of laminitis in the horse.

Acute equine laminitis is almost universally considered as an intoxication of pedal localization. According to the conditions surrounding its appearance, bacterial alimentary toxins and waste products of muscular work or cellular changes, which have disturbed the vasomotor phenomena of the keratogenous membrane, without inflammatory or infectious lesions are suspected. It is only at the end of several days that serious or hemorrhagic collections form. The diversity of etiologic factors in view of the constancy of symptomatology and lesions suggests the existence of a single intermediary factor.

Histamine has vaso-dilatory action which "affects little besides the capillaries, causing in them a sort of acute paralysis; it predominates in the capillaries of the intestine and the extremity of limbs" (Tinel). Moreover, the permeability of the capillaries is increased, this phenomenon being in a certain measure independent of their dilatation (Delherm). There is in this a striking correspondence with congestion of the keratogenous membrane in laminitis.

Then, histamine is a disintegration product of alimentary or tissue albumins (it is produced by the decarboxylization of an amino acid, histidine, which is a constituent of these albumins). It is a very harmful waste product, and very easily liberated in the organism. It may be the single intermediary factor whose existence we have suspected.

Histamine like laminitis, brings on myosis (Delherm, Mintsheff), and an increase in the frequency of cardiac contractions (Delherm, Desliens).

In laminitis there is a disturbance of equilibrium of the autonomous nervous system beginning with a vagotonic crisis and due to heterogenous albumins. This conception is related to peptonic shock, which itself is now compared to histaminic shock: in the conditions of shock, there would be a basic liberation of histamine.

The behavior of arterial pressure is the only inconsistency in this comparison. In acute equine laminitis, the pressure rises (Desliens), while in man histamine brings a lowering of pressure. It is true that important differences in the action of histamine are noted in different species; it is only in man, monkey, and dog that it dilates the small arterioles; according to Perrault (Loeper), it induces hypertension in the herbivores. But Desliens has noted histaminic hypotension in several very important experiments on the horse and also on the cow.

We do not believe it necessary to take this discrepancy too seriously. It may be that besides histamine other substances are formed which themselves have a hypertensive action by vasoconstriction of the arteries and arterioles; but it is not necessary to have recourse to this hypothesis. The vasomotor phenomena are always very complex and there often exist synergetic or antagonistic secondary actions which exceed the primary action.

First, it is recognized that histamine activates the secretion of adrenalin (Tinel, Delherm, Loeper), the hypertensive hormone; by vasoconstriction of the arteries, arterioles, veins and venules.

Then, pain is a very important hypertensive factor, its principal effect being a vasoconstriction of small arteries and arterioles caused especially by medullary and upper reflexes (Desliens). Desliens has even shown that in painful affections, and especially in laminitis, hypertension is in direct relation to the pain. The keratogenous tissue, congested, and on that account compressed between the bone and the rigid horny casing, is certainly the seat of a particularly intense pain. In support of this interpretation, we point to the comparison that can be made between revulsion and laminitis. It is now known that revulsion produces a local liberation of histamine by irritation or alteration of the tegument. But Desliens has clearly shown in his experiments upon the horse that revulsion (counter irritation, blistering, firing) produces a strong hypertension under the influence of pain (Desliens).

Finally, histamine is often found to be liberated, in a particularly intense and crude way in the midst of certain tissues. It may be liberated in the keratogenous tissue in sufficient quantity to engender an intense congestion, without sufficient absorption by the circulation to induce a general vasodilatation of the capillaries thus generating hypotension, if another hypertensive phenomenon, even though

*Chavance, Jean. Histamine theory of acute laminitis in the horse; its treatment by synthetic antilustamines. *Bulletin de L'Académie Vétérinaire de France*, 18:9. (Nov.) 1945.

slight be set in action to resist it and exceed it.

Capillary paralysis has variable consequences depending on the blood pressure which the capillaries have to bear. A strong pressure at their level will distend them extremely, will cause an intense congestion, and may even rupture them.

A weak pressure causes little modification in them. As to that, there will come into play the general arterial pressure, the condition of constriction or dilatation of the local arteries and arterioles, which serve the paralyzed capillary network, the more or less dependent position of the region and the gravitational brake on the return circulation.

In this way, the hormonal or reflex vasoconstriction is, in laminitis, a consequence both unfavorable if generalized and hypertensive, and favorable locally by constriction of the arterioles, which saves the paralyzed capillaries from the rush of blood. The position of the feet aggravates their congestion through gravity, just as, in the triple reaction of Lewis when done on a limb, the red ring due to neuro-histaminic capillary dilatation is exaggerated by a dependent position.

The classic treatments of acute laminitis are interesting to study. Nearly all reveal an agreement with the histaminic theory, if not to combat the cause itself, at least its immediate manifestations. And if no one of them can furnish proof of the theory, their total constitutes a group with a certain deductive value. Their classification may be set up according to their modes of action:

1. Lowering of general hypertension: bleeding purgation, vagotonic alkaloids like pilocarpine and arecoline.

2. Stimulation of return circulation of the limbs: much reduced locomotion.

3. Vasoconstriction of arteries and arterioles whose local effect protects the capillaries: cold baths and showers, adrenalin, cocaine.

4. Elimination, destruction or neutralization of the toxins and the histamine: bleeding, formol. At the end of this study we will add a discussion of the synthesized antihistaminics.

5. Suppression of the neuro-histaminic mechanism of the antidromic and axon reflex capillary vasodilatation: cocainization of the plantar vasculo-nervous bundles.

These observations have led us to consider several procedures capable of being used either alone or together in the histaminic hypothesis of acute laminitis.

(a) Histamine may arise at some point in the organism (intestine in plethoric, laminitis, uterus in laminitis of parturition) and pass by way of the blood to manifest its physiopathologic effects in that tissue most sensitive by its nature and predisposed by its housing in a rigid horny casing and its dependent position, both of which intensify the inflammatory reaction.

(b) The histamine may be liberated locally in the keratogenous tissue itself. By alteration of this tissue due to various factors such as the traumatic shocks of locomotion; the uninterrupted pressure of forced standing on an opposite foot by the other being incapacitated and the more or less specific action on subungual tissue of chemical substances carried by way of the blood (alimentary poisons, microbial toxins). In order that this supposition should agree with the genuine primary congestion of the keratogenous tissue, the alteration of the tissues must be discrete. The alteration may also be localized in a very restricted zone of the membrane and completed for the remainder of the membrane by the two other processes.

(c) The histamine, finally, may be liberated locally by axon reflex following irritation of the keratogenous tissue and of its vessels by the various factors which we have enumerated. It is an attractive conception which takes into account the primary unchanged keratogenous tissue, but which does not seem sufficient in itself to explain all cases of laminitis.

II. Experimental Testing

This histaminic theory of acute laminitis of the horse calls for an experimental test. It is necessary to establish an excess of histamine in the blood of the diseased horse, and at the same time the therapeutic action of the antihistaminics.

The balance of histamine in the blood is very delicate. No study has yet been made to determine for the horse, its normal value and its physiologic and pathologic variations. We were not able to undertake this work.

The working out of a test treatment was better adapted to our means, but to have a demonstration value it must be subject to certain conditions. In laminitis, the light cases, by far the most frequent, often recover spontaneously, and all treatments which supply a slight accessory assistance and which do no harm, are beneficial. To be rigorous, an experiment must exclude these cases, and deal with those serious cases where natural recovery is judged impossible, or as nearly so as this word has meaning in medicine. We had to wait a long time for such a case (observation I) in order to try 2339 R.P. chlorhydrate of N-dimethylamino-ethyl-N-benzylanilin (antergan). A little later (observation II) we tried in a less serious case, 2786 R.P. paraoxy-isophthalate acid of dimethylamino-ethyl-N-meth-

oxy-benzyl-N-amino-pyridine (neo-antergan). These two synthetic product belong to the series of antihistaminic amines and have very pronounced antiallergic properties; neo-antergan seeming to be, at the moment, the most active and the best tolerated member of this series. These two tests have confirmed all the hopes on which we had built.

III. Observations

Observation I.—Acute laminitis of parturition treated by antergan: This case was in a mare, five years of age and of medium build. She experienced painful labor because of the great size of the fetus. Natural delivery, however, was effected.

Laminitis set in 48 hours after delivery and our first visit took place 24 hours later. At that time the case appeared very serious. The mare, with the freedom of a large stable, was fixed on her legs in complete immobility. It took pulling by several men to move her even a little. All four feet were affected. This condition was accompanied by other usual symptoms. Fearing puerperal infection, we did not practice bleeding, and injected 40cc of antigangrene serum. Treatment of the laminitis consisted of an intravenous injection of 30cc of a commercial preparation containing 1.5cc of 40% formaldehyde solution (formalin) and 5cc of a 1 to 1000 solution of adrenalin.

The condition was practically the same 48 hours after this treatment. It was always necessary to pull the animal forcibly to induce it to advance, painfully, a few steps. The feet sweated on their plantar surfaces, and "soles" of straw stuck to them. The treatment should have produced a definite improvement by this time. Its lack of success confirms the gravity of the case. We did not believe that the life of the mare, which had kept her appetite, was in danger. On the contrary, the transition to a chronic condition seemed to us inevitable. In any case, at best it might be thought that no remarkable improvement would develop in the days immediately following. These conditions seemed to us sufficiently severe to make a test of antergan. Forty cubic centimeters of a 2.5% solution containing 1gm of the pure product was injected very slowly in the vein, without any reaction.

All hours and dates which follow refer to this first injection of antergan taken as the starting point.

Seven hours: there is a very definite amelioration, but the condition is still serious; 1gm of the product is injected again.

24 hours: a very important improvement permits the forecast of a cure close at hand. A third and last injection, this one of 0.5gm is made.

48 hours: the animal is practically well. It walks about by itself in the stable, showing barely a slight limp in a foreleg when it makes a sharp turn on it. The feet still sweat a little.

Five days: the animal trots by itself in the stable yard without limping. Eventually the complete recovery is confirmed.

An action so rapid, even though instituted late in so serious a case, seems to us a proof demonstrating the histaminic theory of laminitis and its rational treatment with antergan.

Observation II.—Alimentary laminitis treated by neo-antergan: A draft stallion, 12 years of age, heavy build, medium condition, got loose during the night of the 12-13 June and ate an indeterminate quantity of cracked barley. In the evening of the 15th, it had great difficulty in moving about; this was evidently the onset of laminitis.

On the 16th, at 8 o'clock, our first visit took place. The horse was immobile in its stall, fixed on its four limbs. It could move only with difficulty, with the classic signs of acute laminitis of all four feet. Temperature 38.9° C. From the general point of view, this case was mild. Locally, it seemed that it must evolve slowly (in about eight days, perhaps) toward recovery with some slight risk of distortion of the feet as a consequence. We gave a waiting treatment of an intravenous injection of formol-adrenalin.

On the 16th, at 11 o'clock, having just received the neo-antergan for which we had been waiting for several days, we injected into the jugular 36cc of a 2.5% solution, i.e., 0.9gm of pure product. The horse, which was in the same condition as in the morning, showed no reaction.

On the 16th, at 20 o'clock (8 p.m.), the condition of the patient was transformed. In the stable he rested now on one foot, now on another, no different in appearance from the neighboring animals. The gait was no longer distressing, but the forelegs still took short steps which betrayed the persistence of some pain. Temperature 38° C. A second injection of 0.9gm of neo-antergan was administered.

On the 17th, at 11 o'clock, the general condition was perfect. The horse sought food eagerly, the temperature was 37° C. The gait was almost normal. A third and last injection of 0.6gm of neo-antergan was given. On the 18th, at 8 o'clock the horse walked with a completely normal gait. Subsequently the complete recovery was confirmed.

The interest of this case lies in the rapidity of action of neo-antergan, which in nine hours, produced a considerable improvement, and complete cure in 48 hours.

Contagious Ovine Pneumonia Caused by a Filterable Virus*

By PROFESSOR ZAKI MORCOS
M.V.Sc., F.R.C.V.S., D.V.H. (Eng.),
OSMAN A. ZAKI
M.Sc. (Cairo), B.Sc. Hons. (Liverpool) and
RAGHEB ZAKI
M.V.Sc. (Cairo)

Literature

Friedberger and Frohner (1910) reported an enzootic catarrhal pneumonia in lambs in which the symptoms were fever, pneumonia and dullness of the lungs, usually unilateral. The post-mortem lesions were extensive catarrhal desquamative foci of inflammation sometimes accompanied by caseous necrotic areas. In some cases, pleurisy and purulent pericarditis were met with. The real cause of the disease remained unknown. Cowdry (1925) reported a specific catarrhal pneumonia of sheep occurring in South Africa in which large areas of the lung were edematous and inflamed. The cause of the disease was a virus commonly associated with a small gram-negative bacillus and a gram-positive diplococcus. Henderson (1929) stated that contagious pleuro-pneumonia occurred among goats in Nigeria. The disease caused heavy mortality every year but the cause could not be thoroughly investigated owing to shortage of research officers. Dungal (1931) reported the occurrence of outbreaks of acute infectious pneumonia among sheep in Iceland which was caused by Pasteurella-like organisms. Miesner and Koser (1931) reported that in about 50% of cases of lamb pneumonia in Germany, a specific organism could be detected. Of these organisms the most common was *P. oviseptica* (43%), while *C. pyogenes* and streptococci were occasionally isolated. Walker (1931) reported that contagious pleuropneumonia of goats in Kenya could be experimentally reproduced by injecting the filtered pleural exudate into the chest or trachea of goats. Delpy (1932) reported a enzootic pneumonia among sheep in Persia in which no symptoms attracted attention until a few hours before death. The cause was claimed to be a Pasteurella and an unidentified coliform bacillus. Stylianopoulos (1934) reported pleuropneumonia among goats in Greece which was caused by a virus localized in the affected lungs and pleural exudate. Doukaloff (1935) reported that infectious pleuropneumonia in goats caused considerable losses in Russia. Artificial infection could be carried out by inoculating liver or spleen emulsions with or without filtration, proving thus that the cause was a filterable virus. Gilbert (1937) reported an infectious pleuropneumonia among goats in Palestine which was communicable to sheep. The mortality amounted from 60 to 80% in goats and 40 to 50% in sheep. The cause was stated to be a filterable virus. Blakemore and Bosworth (1938) reported the occurrence of "Jaagziekte" disease in a flock of sheep in Norfolk in which the characteristic symptom was a clear serous discharge from the nostrils. The post-mortem lesions were confined to the lungs and bronchi. The disease was also encountered in sheep in England. Gislason, Dungal and Taylor (1938) reported the introduction of a new disease in sheep into Iceland characterized by adenomatosis in the lungs. Nothing definite was known regarding the etiology of the disease. A gram-negative capsulated diplococcus resembling the organism of contagious pneumonia and a Streptococcus were isolated from typical cases but were not infective to other animals on artificial inoculation. Taylor (1938) failed to reproduce adenomatosis experimentally. He believed that "Jaagziekte" disease and adenomatosis were the same and that it was not uncommon among British sheep. Baumann (1938) stated that infectious pleuropneumonia in goats had been recognized in Algiers, Germany, Italy and Greece and that the etiological agent was said to be a filterable virus although a variety of bacteria, especially Pasteurella, were isolated in advanced cases. Bosworth, Montgomerie and Glover (1938) reported outbreaks of enzootic pneumonia in sheep in North Wales and East Anglia. Gram-negative coco-bacilli not unlike Pasteurella were isolated from the hepatized lungs. Nilmi (1938) isolated *P. oviseptica, C. pyogenes* and a small gram-positive bacillus in enzootics occurring among lambs in Japan. Marsh (1938) reported an acute bacterial pneumonia in lambs which caused serious losses among flocks of Rocky Mountain Bighorn Sheep. Pasteurella associated with *C. pyogenes* proved responsible for the outbreak. Langman (1941)

*From the department of Bacteriology, Veterinary College, Fouad I University, Cairo.

isolated *P. oviseptica*, beta- Streptococcus, diplococci, *Staph. aureous, Staph. albus, E. coli* and *N. catarrhalis*-like organisms from nine cases of sheep pneumonia in America.

History

During the winter of 1942-1943, somewhere in the open in the desert surrounding the Pyramids of Gieza, some hundred fully grown, Sudanese sheep were imported by the British Army for the supply of the Indian Troops in Egypt. An enzootic disease was reported to this department by the R.A.V.C. authorities. The spotting symptoms of the disease were intermittent fever, extreme, rapid emaciation, nasal discharge of variable intensity and, most prominent, were small blisters not unlike those of pox around the lips and rarely on the abdomen and thighs; none were seen under the tail. Later, the vesicles broke leaving ulcers. Gastrointestinal disturbances were not uncommon. The post-mortem lesions were more or less confined to the chest cavity. The lungs were congested and pneumonia as well as pleurisy were met with. The abdominal organs were congested and swollen.

In the winter of the following year, a similar outbreak occurred among the sheep of the Faculty of Agriculture at Gieza in which 50% of the animals died. Specimens were brought in to this department for investigation. The symptoms were rise of temperature, loss of appetite, depression and nasal discharge. Some times there was a short dry cough and edema of the eyelids with watery discharge. Later, the nasal discharge became mucopurulent and the respiration hurried and labored. Constipation followed by diarrhea was a prominent symptom. In some cases there were vesicles on the gums and lips which ruptured leaving raised ulcers. The disease was of short duration in lambs and some died suddenly without showing symptoms. In aged sheep, however, the disease took a subacute and sometimes a chronic course, which lasted from 15 to 20 days.

The post-mortem lesions were chiefly confined to the lungs. In peracute cases there was severe congestion and edema of the lungs. In acute cases consolidated areas of varying size, dark red or greyish in color were seen. Sometimes caseous and necrotic lesions were met with. In subacute and chronic cases fibrinous pneumonia with gangrenous areas containing mucopurulent fetid discharge was a constant feature. In some cases small caseated nodules containing cheesy-like material were scattered in the lungs. The pleura of the affected parts showed fibrinous inflammation with adhesion. The pericardium was inflamed and often contained blood-tinged or purulent material. The liver, spleen and kidneys were congested and slightly enlarged. The gall-bladder was distended with thin greenish bile. The gastrointestinal tract was deeply inflamed.

Experimental

The blister-contents on some of the affected sheep were aseptically collected and diluted five times with sterile saline. This was inoculated intradermally into guinea pigs and rabbits. After three days the seat of inoculation was emulsified in saline and reinoculated intradermally into a series of guinea pigs and rabbits, but neither local nor secondary vesicles were reproduced. Thus excluding both foot-and-mouth disease and pox viruses.

1.—Blood from dead sheep was diluted 20 times in saline. Spleen and liver were separately emulsified and similarly diluted. Each was then filtered through Seitz filter (five inches in diameter), the filtrate controlled and inoculated intraperitoneally into rabbits, guinea pigs, white rats, mice and pigeons. Five animals of each were used. Only the rabbits were found susceptible and these died within 10 days after inoculation. Post-mortem examination revealed congestion of the lungs and severe gastroenteritis. The liver, spleen and kidneys were slightly congested. Blood films and culture were negative for microorganisms.

Blood of the dead rabbits diluted and filtered as before, was again inoculated into rabbits which died after five days with similar post-mortem findings. Further passage of the virus did not further exhalt its virulence for rabbits.

II.—Blood filtrate was inoculated intramuscularly into lambs, about two months old. These showed intermittent rise of temperature, intestinal and pneumonic disturbances and died in two weeks. The post-mortem lesions were catarrhal pneumonia and gastroenteritis.

III.—In about 60% of the examined lungs especially those of the chronic cases, a variety of microorganisms, secondary to the virus, were isolated. Bacteriological investigation identified the following organisms:

P. oviseptica.—This organism possessed the typical morphological and cultural characters of Pasteurella. Gelatin was not liquefied. Milk remained unaltered. Indol was produced. Gas was not produced from any of the carbohydrates. Acid was formed in glucose, maltose and mannite. It proved pathogenic to guinea pigs and rabbits which died in 24 hours after inoculation.

Pasteurella-like organisms.—These were gram-negative cocco-bacilli which morphologically resembled Pasteurella. They, however, grew more luxuriantly on ordinary culture media. No gas was produced from the ferment-

able carbohydrates. Milk was not changed and gelatin not affected. Indol was not produced. The biochemical reactions were variable. Some strains fermented maltose and saccharose only; while others fermented glucose, maltose, saccharose and mannite. They proved non-pathogenic to rabbits and guinea pigs.

Coliform bacilli.—These organisms most probably belonged to the anaerogenic type of *E. coli.* They were gram-negative cocco-bacilli and grew well on ordinary media. They fermented lactose, glucose, maltose, saccharose and mannite. Milk was acidified and clotted. Indol was produced. Gelatin was not affected. The organisms were non-pathogenic to laboratory animals.

Corynebacterium pyogenes.—This organism was invariably isolated from the suppurative lesions of the lungs. It was gram-positive and very pleomorphic, the coccal forms most predominant. The best growth was obtained on serum or blood agar. At first the colonies were minute, but attained the diameter of about 2mm after a few days incubation at 37° C. The organism was hemolytic and liquefied gelatin and coagulated serum. It fermented lactose, glucose, maltose and saccharose with the production of acid only. Mannite was fermented after seven days. Milk was first turned acid and then clotted after 48 hours. The organism was not pathogenic to either rabbits or guinea pigs.

Immunization

Two vaccines were prepared for immunizing purposes:

1—*Vaccine prepared from rabbits.*—The spleen and liver of infected rabbits were collected at the point of death and emulsified in saline containing 1:1000 formalin in the proportion of 1gm of tissue to 30cc of formalized saline. The emulsion was then strained through sterile gauze and placed in sterile rubber capped bottles. The vaccine was kept at room temperature for seven days before use; frequent shaking was adopted.

The vaccine was tested on five young Egyptian bred rabbits, of an average weight of 700gm. Each rabbit was inoculated with 1cc of the vaccine subcutaneously. Three weeks later, each animal was injected with a test dose of 0.25cc virulent sheep blood filtrate. An equal number of unvaccinated rabbits were similarly treated to serve as controls. All the vaccinated rabbits survived while all the controls died within seven days with the same post-mortem picture as was shown previously.

2—*Vaccine prepared from sheep.*—In preparing this vaccine the liver and spleen of sheep dying from the disease were used, the same technic being adopted as before. It was then kindly tested by Doctor Rida on sheep at the Faculty of Agriculture where the disease still existed. Twenty lambs were inoculated twice with a week interval, the first dose being 2cc and the second 5cc, injected subcutaneously. Three weeks later the vaccinated animals and four unvaccinated controls were injected with a test dose of 0.25cc virulent blood filtrate. Eighteen of the vaccinated animals survived; two died from the disease. Only two of the unvaccinated controls died with the usual symptoms and post-mortem appearance.

Another lot of 30 sheep were similarly vaccinated and left in contact with naturally infected animals. Of these two died and 28 survived.

In the interpretation of these results it appears that the vaccine had a good immunizing power. The survival of the two unvaccinated control sheep was most likely due to immunity acquired from natural sublethal infection and recovery or to inherited resistance.

Summary

1. A filterable virus has been found to cause contagious pneumonia in sheep. The virus was present in the blood and internal organs.

2. The virus was pathogenic to rabbits, but not to cavies, white rats, mice nor pigeons, on experimental inoculation.

3. Secondary organisms were isolated which played an important part in complicating the disease.

4. Formalized tissue vaccine prepared from liver and spleen gave promising results.

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A Practitioner's View of Brucellosis in Dairy Cattle*

By S. L. STEWART
Olathe, Kansas

INFECTIOUS abortion in dairy cattle have been known since the days of Jacob, the resourceful son of Isaac, yet we are still fighting it. Nearly 150 years ago White[1] described it accurately; recognizing that the disease is communicable. Clater[2] 1810 stated that it occurs as an epidemic in some herds.

Brucellosis has been known by 10 or a dozen different names, over the last 50 years, but the history of names need not interest a practitioner especially at this time, as the profession has finally settled on brucellosis.

Brucella abortions are more or less common throughout this country, and are reported to be common throughout the world. Basing our judgment upon these indications we must conclude the disease is on the march, but veterinarians should never run up the white flag on the fight to clean up brucellosis, *because it can be done,* and it is not the American veterinary way to let it go undone.

Brucellosis seems to be only slightly contagious in some herds. In such herds its invasion can hardly be called an outbreak. In other herds the infection is highly contagious and its introduction is signalized by many abortions at different stages of pregnancy.

The difference may be accounted for by one or more of at least four factors, which come under two major classifications.

1. In herds where the infection is mild, either of two factors may be responsible. (a) The organisms are of low virulence, and barely capable of producing abortions or (b) the cattle in the herd may possess a strong natural or acquired resistance to the invading strain of Brucella.

2. In a herd where abortions come as a scourge, and are followed by a high percentage of retained afterbirths and mastitis, one is justified in concluding (a) the organisms are of a highly virulent strain, very infective and capable of producing a storm of abortions, or (b) the animals in the herd are highly susceptible to that particular strain of Brucella that has invaded it.

This characteristic of varying virulence among different strains of Brucella organisms is stated by Breed[3] as follows: "The presence of Brucella infection in a herd should never be regarded lightly. There is always the possibility of mild infection in one herd flaring into a disastrous outbreak when introduced into another herd, or even in the same herd, perhaps, by passing serially through several highly susceptible animals. It has been shown by experimental work and also in field, that not all strains of *Brucella abortus* have the same antigenic or immunizing properties, nor do they all possess the same degree of virulence." Herds have been seen where only one or two abortions occurred during a whole year, and then suddenly many abortions occurred within a few months.

While we rightly attribute the cause of abortions to *Brucella abortus* infection, we should not lose sight of the fact that a herd may be infected with a strain of low virulence and react to the agglutination test, and at the same time be suffering from a deficiency; probably both mineral and vitamin to an extent that the deficiency is causing many abortions. Here is a case where the practitioner can go out on a limb. Every reactor can be eliminated from such a herd without affecting the incidence of abortions, if the feeding practices remain unchanged.

The matter of deficiency is made more complex by the fact that it differs in different sections of the country and even on different farms in the same community and in different seasons. It depends upon soil, climate, the crops and farm practices.

The veterinarian in dairy cattle practice where the cows are highly bred, and highly fed for maximum milk production, has an opportunity to observe many significant things in brucellosis and also in deficiency diseases. It is his privilege to study the herd and its individual members. The knowledge so acquired serves him well when trouble comes and places him in position to make correct decisions as to how best to handle the herd. Herd management is of great importance, and no one else can equal the veterinarian, who is intimately acquainted with the herd, in giving the owner good advice on herd management.

Some very peculiar things have been observed in practice. The phenomena of brucellosis have caused a great deal of misunderstanding and a lot of trouble. A few case re-

*Some parts of this discussion were presented at the annual meeting of the Indiana Veterinary Medical Association, Indianapolis, Jan. 10, 1946.

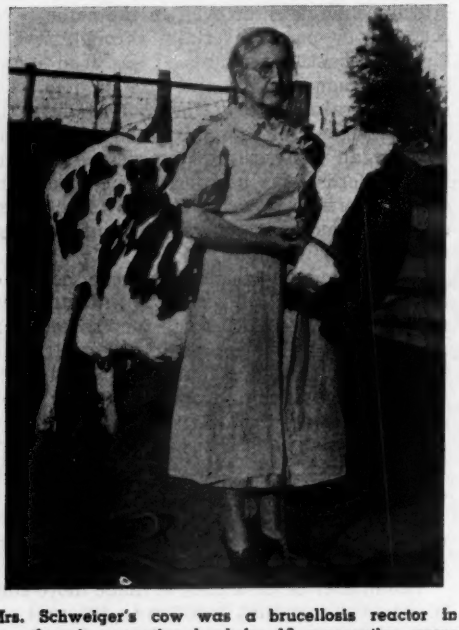
[1] White, James. A Compendious Dictionary of the Veterinary Art, 1801. Reprinted, 1817.

[2] Clater, Francis. Every Man His Own Cattle Doctor. 1810. Reprinted, 1815.

[3] Breed, Frank.

ports may be cited to illustrate some of the misunderstandings about handling cases of this disease.

No. 1. (Schweiger cow)—A 16-year-old cow was bought by a client of mine when a three-year-old and placed in an abortion-free herd. She calved within a few days and retained her afterbirth. She was then given the agglutination test for brucellosis and reacted positively in three dilutions. This cow possessed unusual



Mrs. Schweiger's cow was a brucellosis reactor in an otherwise negative herd for 12 consecutive years following her purchase as a three-year-old. During that time she produced 12 normal calves and some 160,000 pounds of milk containing approximately 6,000 pounds butter fat

milk producing qualities and for that reason was retained in the herd. She was vaccinated[4] with live culture vaccine at the time the afterbirth was removed, and revaccinated when known to be pregnant. She was blood tested every six months as long as she lived or 26 times thereafter and was positive on every test. She remained in this negative herd for 13 years, up to the time she died, at 16 years of age, was still a positive reactor. She produced 12, normal, living calves and her heifer calves were all retained in the herd and all proved negative on every six-month test. In her life time she produced approximately 160,000 pounds, or about $3000.00 worth of milk and 12 good calves. It cost $5.00 to remove her afterbirth and $4.00 to vaccinate her. She spread no brucellosis in the herd during 13 years of continuous association with negative cows.

This is a report on only one cow, but the case is unusual only in the great age to which this cow remained a good producer. Everyone in cattle practice has seen cows that were regular breeders and good producers that reacted to the brucellosis test year after year and yet were never suspected of infecting others. Many cows as good as this one and probably as valuable to the owner, have been condemned on the blood test and sent to slaughter. I now confess that I have been guilty, many times, of sending valuable cows to slaughter.

No. 2. (Zhwisler cow)—A Jersey, the family cow for three adults and three children: This cow aborted at about the eighth month of gestation and retained the afterbirth. The agglutination test showed positive. She aborted her next calf and retained the afterbirth. The owner then consented to vaccination and she was given 10cc of abortion vaccine at the time the afterbirth was removed and another 10cc dose before she was bred, 90 days after the abortion. She conceived at the first mating, delivered a normal calf and expelled the afterbirth. About one year from the date of her last abortion, or soon after delivering a live calf, she was negative to the agglutination test, and was sold into a negative Jersey herd. She was blood tested every six months thereafter, and continued negative. Her heifer calves were retained in the herd and they also were negative to the tests at six-month intervals. The important feature of this report is, that the cow aborted two calves; was then vaccinated and in 12 months was negative and remained negative and bred regularly.

No. 3 (Harness herd)—This was a small herd of mixed breed dairy cows in which there was one abortion in 1942. In February, 1943, abortions began to occur in rapid succession. The agglutination test showed 50% of the herd to be positive reactors. A 10cc dose of vaccine was given to all females in the herd, but the abortions continued. In April the entire herd was revaccinated with a 10cc dose; but abortions continued. In May the herd was vaccinated for the third time using a 10cc dose, and still abortions continued. Then by reminiscing a little, I remembered the time when I required all vaccinated herds to be fed vitamineral for at least 90 days immediately following vaccination. Vimogen was recommended for this herd and was given on feed twice a day for 120 days. Not a cow in that herd has aborted from that day to this, a period of nearly two years. I am convinced

[4] Whenever vaccination is referred to in this discussion intramusclar injection of 10cc of strain 19 vaccine is meant except in calves where the dose is 6cc.

that even though 50% of this herd was positive to the agglutination test the abortions were caused by a nutritional deficiency and not by the *Brucella abortus*. Some veterinarians that I know would not have been so easily convinced. They would want the mineral withheld from half the herd for controls and would want to repeat the experiment a half-dozen times, which is all very well, but I thought I had already displayed quite sufficient obtuseness in the matter.

No. 4. (Moore)—A storm of Brucella abortions in a small registered Jersey herd resulted in 25% of the cows aborting within a very short period. In this case the disease was thought to have been introduced into the herd by a fine two-year-old bull that had been sold into two herds previously but was not tested before entering this herd.

The herd was vaccinated with good results; only one three-year-old cow, which had aborted before vaccination, aborted afterward. But she continued to be a source of annoyance. She aborted her second calf five months after vaccination. She was revaccinated while open. She aborted her third calf at five month pregnant, making three abortions for her in about 13 months. Before her third abortion she developed an unusually severe and acute mastitis with severe mammary lymph glands involvement, one quarter of the udder was soon lost, and the cow came very near dying. After she recovered from mastitis, she was sold on the market for slaughter.

By all the usual criteria this cow suffered from acute brucellosis regardless of the fact that she was vaccinated twice with a 10cc dose each time. Apparently she did not immunize. She was thought to still be on fire with acute infection at the time she was sold but this was not proved. Each time she aborted, she required help to deliver the fetus, and the afterbirth had to be removed, manually, which every time showed the characteristic pus and diseased cotytedons of Brucella abortion. She was a dangerous cow. Each time I delivered her and removed the afterbirth, I contracted a very acute allergy regardless of allergy preventive treatment.

The source of brucellosis infection in a herd is not always known. Usually it is a new cow —maybe a new bull—or a neighborhood bull, or infected cows brought into the herd. Surface drainage may have something to do with spreading disease. Maybe contaminated straw kicked from a commercial stock truck occasionally introduces it, but I hold that the living animal and the decidua from an aborting one are the important factors. Positive cases sometimes show up in negative herds without trace of the source of the infection and no other positive reactors occur in the herd it may be for years.

In my estimation it is far better to prevent than it is to cure disease. But a practitioner has a poor break in controlling brucellosis in dairy cattle, except in individual herds. Dairymen are just not sold on the prevention of this disease. Nevertheless the veterinarian must do for his client the very best he can to pull him out of a bad situation, after his herd becomes infected. The practitioner who is in

A practitioner in the field every day of the year who knows the herd is in a better position than anyone else to handle a brucellosis outbreak in that herd

the field 365 days in the year, is acquainted with conditions at the dairy farm and usually well acquainted with the herd, and herd management. He is in a far better position to handle any brucellosis outbreak than anyone unacquainted with conditions. Furthermore, he must handle it successfully or quit. He holds his clientele by rendering satisfactory service. He can't render a satisfactory or any other kind of service in a herd that is eradicated or for a dairyman who has been forced to go out of business. But he doesn't fail if management of the herd is left to his discretion.

There are three things to be considered in the matter of brucellosis. (1) It is an animal disease problem. (2) It is a public health problem and (3) it is a personal problem to the veterinarian because nearly all who do a dairy practice develop a brucellosis allergy.

The disease in animals is strictly a veterinary problem. The desideratum is eradication from

every infected herd. Common sense demands that, if possible, the eradication be accomplished without bankrupting the owner. I mention this obvious fact only because it has often been forgotten. Eradication of brucellosis should be done with the minimum loss to the dairyman and with the least upset to the dairy cattle industry which the circumstances permit. It is the writer's belief that it is possible to eradicate bovine brucellosis from the United States in five years and that it should be done in twice that period even allowing for unforeseen hindrances.

The public health problem that brucellosis poses would be solved by eradication of the disease in animals. However, in the meantime the problem is a very real one and is increasing in seriousness year by year. Public health authorities have been very lenient with unproductive efforts to control this disease for the past decade. There is evidence that their patience is wearing thin in spots. Unless we make more progress in the future than we have in the past we may find some stringent regulations imposed on the sale of milk from infected herds. Veterinarians should cooperate fully with public health authorities and lend them every assistance possible in their efforts to limit the spread of the disease to persons.

Brucellosis allergy affects many persons. Veterinarians are more exposed to brucella infection and brucella allergy than any one else except certain packing house employees, for it is the veterinarian who must care for sick animals.

Few veterinarians suffer from undulant fever, notwithstanding dairy practitioners contact reacting cows daily. In time almost 100% of them become allergic to brucellosis. While the allergy does not cause death and seldom if ever puts a veterinarian in a hospital, yet it is so disagreeable and annoying that some veterinarians have retired from practice rather than put up with it. And these veterinarians, because of their experience, are the ones most needed in dairy practice and especially in helping to control and eradicate brucellosis.

The writer has seen a man who was so allergic to brucellosis that exposure caused sores and red splotches almost covering his arms, neck, face, genital organs, and much of his body. Another case was a five-year-old girl, who, according to her physician, was so allergic to cows milk that after drinking it she would become weak and nauseated until vomition emptied her stomach after which she would feel well, until she drank cows milk again. The little girl thrived on goats milk, however. The herd of cows, from which the family obtained their milk, was badly infected with *Brucella abortus*, and many abortions had occurred in it. There is a question in my mind as to whether this girl was allergic to milk or to Brucella organisms and their products in the milk. Abortions were so frequent in this herd that the dairyman was forced either get rid of abortions or quit the dairy business. He chose vaccination and now, four months later, no more cows have aborted and his mastitis situation has materially ameliorated.

Dr. I. Forest Huddleston has done a remarkable piece of work on Brucella allergy. Every veterinarian should read his two bulletins: Brucella Allergy in Veterinarians and Prevention of Brucella Allergy in Veterinarians.

The prevention and treatment for allergy that I have used for the last year with some measure of success is as follows: Immediately after attending an obstetric case—either delivery or removal of the afterbirth:

1. I wash my arms thoroughly with soap and water.

2. I rinse my arms with a solution of one ounce of dilute hydrochloric acid in one gallon of water, and do not wipe them but allow them to air dry.

3. I spread an immune serum evenly over the arms and allow it to remain for two or three minutes.

4. I rinse off the serum with the dilute hydrochloric acid solution. This leaves the skin acid.

5. I cover the arms with talcum powder for its drying effect. This has proved effective in most cases in preventing Brucella allergy, but has completely failed following contact with some infected cows.

There are only two ways of controlling or eliminating brucellosis from our dairy herds.

1. By test and slaughter.

2. By vaccination.

If coöperation of the U. S. Department of Agriculture, the federal Bureau of Animal Industry, the livestock sanitary authorities of the 48 states, the practicing veterinarians in all the states and the cattle industry could be perfected my prediction is, the job could be completed in from five to 10 years. The test and slaughter method has a place and should be available to cattle owners who want it. However, we must not lose sight of the fact that a positive agglutination reaction does not differentiate infected animals from those that have been vaccinated or have recovered from an attack of the disease or have otherwise acquired an immunity.

In my judgment the biggest blunder ever made in brucellosis control was removal of the restrictions on the sale of brucellosis vaccine and turning it loose to the general public to be used promiscuously, right or wrong. Now it cannot be told by testing whether an animal

is infected or a vaccinated positive or vaccinated negative. It is my judgment based upon a quarter of a century of experience that brucellosis will be controlled only by vaccination and herd management. I believe in the vaccination of all infected herds, as vaccination gives good results in every herd, where good herd management is followed.

After using live culture[5] vaccine since 1922, in the vaccination of entire herds and individuals in other herds, I entertain no doubt that brucellosis can be controlled by vaccination. The period of greatest susceptibility appears to be between the 30th and 90th day of gestation. The *Brucella abortus* appears to be at the peak of activity during this period and, perhaps strangely, vaccination at this time gives the largest measure of success. Some cows possess so much resistance to the organism when neither lactating nor pregnant that it is doubtful if vaccination has much effect on them at such times.

Vaccination of pregnant cows will not cause abortion, but some pregnant cows will abort in spite of vaccination. However, I have been in practice too long to expect any vaccine to immunize every time, be it hog cholera vaccine, rabies vaccine, encephalomyelitis vaccine, blackleg vaccine or any other. An abortion once in a hundred times after vaccination might scare a research man out of a year's growth but a practitioner would be naive indeed to be disturbed by it.

Nor must we forget there are other causes of enzootic abortion than brucellosis. And as for sporadic abortion—well some cows, and other females, too, seem born to abort.

The loss of milk and calves in a nonvaccinated, infected herd is often appalling and, over a protracted period, cannot be endured by the dairyman and still stay in business.

In the foregoing I have indicated what I believe is necessary to suppress brucellosis in all the dairy herds of this country and stop material losses from it within the next decade. To recapitulate it includes:

1. A mutually coöperative arrangement among all veterinary practitioners who desire to participate in the work, the state livestock sanitary authorities and the Federal Bureau of Animal Industry.

2. Whole hearted support of the project by the cattle industry.

3. Complete control of the interstate distribution of brucellosis vaccine by the federal Bureau of Animal Industry and complete control of its intrastate use by the state livestock sanitary authorities, the effect of this dual control being to limit its application to official use by veterinarians.

4. Vaccinate all calves (both bulls and heifers) in all dairy herds between the ages of eight and 12 months. Vaccinate all adult cows in infected herds once and revaccinate any that abort. Of the present vaccine, use 6cc for the calves and 10cc for adult animals.

5. Keep complete records of all vaccinated animals.

For political reasons the foregoing cannot be put into effect all at once but if it is kept in mind as an ideal to work toward we shall get much further than by the haphazard methods of the past.

The principal opposition to the plan would likely be:

1. Veterinarians not participating in the work would object to regulations taking away their privilege of using brucellosis vaccine. They would waive their objections when shown the restriction was for the benefit of the dairy industry.

2. Farm organization would oppose the same restriction but in time could be won over as was the case with tuberculin.

3. Some owners of "clean" herds would object to the vaccination of their young stock. This opposition would not be as serious as it at first appears. Thousands of them have already taken this step and others would embrace it as their own or their neighbor's herds become reinfected.

4. Manufacturers of vaccine who now profit by the promiscuous sale of it to the general public would oppose the restrictive regulations with all their resources. They would just have to be fought.

It will bear repeating that the control and eradication of brucellosis requires the *coöperative* efforts of four groups—the federal Bureau of Animal Industry, the state livestock regulatory authorities, veterinary practitioners and the livestock owners. Regulations under which the work is prosecuted should be the result of their combined wisdom. It is a cardinal principal that the condemnation of a valuable reacting cow when she can be rendered serviceable and safe by vaccination cannot be justified, morally or economically. Regulations that, directly or *indirectly*, require the condemnation and slaughter of such cows are wrong.

It seems to me there should be only one, instead of several plans, for the control and eradication of brucellosis. Enrolling a herd under the plan should be voluntary on the part of the owner, but once enrolled, every female animal and the bulls in the herd and all accessions by birth or purchase should be subject to the brucellosis regulations so long

[5] In the beginning strain 19 vaccine was not used for obvious reason.

as the herd remains in the plan. Most of those herds will be free from the disease, and the positive reactors will have been replaced, in four or five years from the beginning of herd control, but the herd should remain under supervision as a protection against reinfection. Complying with the regulations would, of course, entail some inconvenience but no real hardship to herd owners, and they would be repaid well for their efforts. From my experience I believe they will agree, 100% willingly, to reasonable restrictions if shown that such are necessary.

The federal Bureau of Animal Industry should be the final authority in the project, with full control of the interstate movement of animals from herds enrolled in the plan. State authorities should not set up different regulations for the importation of animals. To do so destroys any chance of uniform requirements. The state livestock sanitary authorities should control and supervise the work within the states, through the veterinary practitioners. The immediate supervision of the herd enrolled in the brucellosis control and eradication project should be vested in a local veterinary practitioner of the cattle owner's selection, insofar as that is practicable.

Any removal of cattle from a herd enrolled in the brucellosis control and eradication project should be on permit from the veterinarian in charge; such permits being revocable for cause by the state or federal authorities. The herd owner should keep a record of his herd on prescribed forms which should be inspected periodically by the veterinarian in charge of the herd, and approved or corrected and signed by both him and the herd owner. When an animal is sold into another herd a transcript of her record should go to her new owner. Drawing samples for the blood test should be performed by the veterinarian in charge of the herd; whether he, or a state laboratory, tests the samples is perhaps unimportant. Personally, I prefer to test my own samples; matters arise frequently that are difficult to explain to a laboratory technician having no knowledge of the herd. If every state maintained a laboratory for testing and the regulations were the same in all states, there would be advantages in having the testing done in a central laboratory. In any case, the blood test should be for the purpose of supplying the veterinarian in charge of the herd with information and should *not* be considered as supplying a diagnosis for him.

The responsibility for the selection of animal replacements or for additions to other herds will not be misplaced if put upon the local practitioner who is better able than anyone else to make the selection and must live with his mistakes if he makes them.

The objective is, or should be, to eradicate bovine brucellosis from the whole of the United States in the shortest practical time with the least possible cost to the owners of cattle consistent with rapid progress. The regulations should be designed solely to accomplish this. There are four factors involved—the federal Bureau of Animal Industry, the state livestock sanitary authorities, veterinary practitioners and cattle owners. Each should work in complete coöperation with each of the others. Each has his sphere of action and his responsibilities. None should presume to omnipotence in the field of another. It would be farcical for me to assume responsibility for interstate shipment of animals or supervision of the manufacture of biological products and it is equally absurd for one to sit in Washington (or Topeka) and assume responsibility for the supervision of a herd in my territory that I have visited perhaps weekly for 20 years and know more about in a minute than any absent administrator can ever learn by studying reports. We have a job to do and it will require the best efforts of all four of us.

Brucellosis

Undulant fever, or human brucellosis, is an infectious disease of world-wide distribution. Primarily, it affects cows, goats and hogs, however, under some circumstances, it is transmissable from animal to man. It has probably been in existence for centuries but it is only within the past two decades that it has been recognized as a serious public health problem in the United States. A few cases were discovered in the Southwest as far back as 1910. Today, some 4,000 cases are being reported annually. In Iowa the incidence is 5.9 per 100,000 population.

Undulant fever is difficult to recognize, especially in its chronic form. Acute cases produce symptoms not unlike those of influenza. In a diagnosis of human brucellosis, the past history of the patient, particularly his or her occupation, is most important. In all cases where there has been close contact with aborting cattle, goats or swine, or where there has been a possible consumption of unpasteurized dairy products, the presence of human brucellosis should be considered. The most important clinical diagnostic signs of the acute form of the disease are weakness, a rise in body temperature in the afternoon and evening, severe chills and drenching night sweats, and all desire for food. This chain of events occurs daily, and produces loss of weight and secondary anemia.—*I. Forest Huddleston.*

Clinical Reports

Foxtails in the Eye

A Pekingese was brought to the hospital on November 1st with a severely affected right eye. The eye had been sore for two or three months and no treatment had helped it. The conjunctival sac was full of pus, the conjunctiva much congested and an ulcer had formed on the cornea.

I noticed a small opening near the medial canthus from which pus could be expelled by pressure upon the eyeball.

The dog was given 2cc of phenobarbital sodium and the eye was examined more carefully.

The small opening near the medial canthus turned out to be about ⅜ of an inch in diameter. With forceps holding the upper and lower eyelids apart, another pair of forceps was inserted in the opening and a foxtail removed. The forceps was inserted again and another foxtail removed.

All in all, eight foxtails were removed from that opening.

The position in which the foxtails lay led me to believe they had entered elsewhere than from the eye.

Examination of the mouth revealed a large ulcer on the cheek just lateral to the right canine tooth. A small opening was found and two more foxtails removed.

A flexible probe inserted in the opening at the medial canthus of the eye came out by the tooth. The tract was flushed with a mild antiseptic solution and sulfathiazole ointment applied to the eye.

The next day, the quantity of pus was much reduced and the eye not so sensitive to touch. Ointment was again applied to the eye and on the day following. The ulcer also was treated. The dog went home the following day feeling much better.

A week later, the owner brought the dog back and the eye was almost free of pus. An injection of sterile milk was given and ointment applied to the eye as before.

Two more injections of sterile milk were given one week apart. At the time of the last injection the eye was almost normal.

I assume the foxtails entered the buccal surface from the mouth, worked their way through the tissues and became lodged against the orbital part of the frontal bone and also against the lacrimal bone.

JOE RIDGWAY

Ventura, Calif.

Delivery of Bovine Monster—Schistocorunus Reflexus

December 25th at 8:00 I received a call from A. T. Pettit at Darlington, Mo. He reported that he had a cow trying to deliver a calf which was dead and (as determined by a neighbor who had volunteered to help) "was rolled up like a football."

Upon making an examination I could appreciate the neighbor's statement: "There's something that isn't right in there."

The liver and intestines of the fetus were hanging from the cow's vulva and the stomach lay in the vagina. These organs together with the lungs were pulled loose from their attachment to the fetus, and removed to facilitate further examination.

The ventral surfaces of the thoracic and abdominal cavities were open, and the ribs and limbs were deflected out and back over the spine.

In the lumbar region the spine was curved abruptly dorso-laterally almost 180 degrees.

Two legs were located parallel to the head in the normal manner except that they were above instead of below the head. A third leg protruded at approximately 45 degrees from the others. All joints were ankylosed.

Using a fetatome the protruding leg was severed from the body and removed. It proved to be the right, rear leg including the tail and about three fourths of the bony pelvis and integument.

As the cow was large and had a roomy pelvis the remaining portion of the fetus was removed with slight traction.

The amputated section was placed back as near possible its original position and the accompanying snapshot was taken.

The left front leg was missing and, due to the dorso-lateral curvature of the spine, the left rear leg was forward and the skin covering it was continuous with that of the head and neck. The right rear leg extended from the left as shown and the right front leg was

in its normal position except, due to the deflection, it was upside down.

No external genitals were present, nor could the sex be determined by gross examination of the internal genital as they were rudimentary and incomplete.

No attempt was made to show the thoracic and abdominal organs in the photograph; as the owner's dog was discovered making his evening meal on them when we had completed the delivery.

The circulation of the fetus was apparently complete and the heart functioning until the time of attempted parturition; as there was no decomposition and full development was indicated by presence of incisor teeth in the lower jaw, and the hooves were developed to normal consistency of a full term calf.

The cow was a seven-year-old, cross-bred Holstein and Angus weighing approximately 1350 lbs. She had previously delivered three normal calves; having milked continually for two years between the second and third calves. The owner explained this by saying: "We were living in town and I could never find a bull at the right time." This pregnancy was by a Hereford bull.

The cow made an uneventful recovery and at present is giving her normal flow of milk.

P. E. KIMBALL

Stanberry, Mo.

"X" Disease

The following report is concerned with a line-bred, Hereford herd of 30 cows, one bull, and 50 calves and yearlings. No new animals (except calves born on the ranch) had been added to this herd for three years. The animals were much too fat.

This past summer was rather dry during August and this herd was on Sudan pasture and had in addition plenty of feed in the feed trough. Rains began in September and the Sudan grass developed many suckers. About this time these cows' eyes began to water excessively. Their noses became very dry and crusty with a profuse mucus discharge. They also began to abort at from four months to full term pregnancy. The skin of all the cows that aborted and of the bull became very thick, dry and covered with scurf. The coat was dead. The skin in the crotch was hard, dry and wrinkled. These animals were hypersensitive to touching of the skin. The mucous membrane became very pale. They lost weight extremely rapidly. This herd bull lost 700 to 800 pounds in less than a month. The cows did not lose weight quite so fast.

A fecal examination revealed the animals to be heavily parasitized. The animals were tested but no evidence of brucellosis was found. A blood sample was taken from each animal. The whole herd was given phenothiazine.

Every cow that aborted retained the afterbirth, not one of which could be removed at first trial. Four ounces of sulfanilamide and urea were placed in the uterus. Stilbestrol, 25mg, was injected hypodermically. After the lapse of 24 hours a second attempt was made to remove the afterbirth. In all but two cows it was removed easily upon the second attempt. Both of these cows died in spite of sulfanilamide, stilbestrol, and urea capsules placed in the uterus.

All the animals showing this skin condition were placed in the barn out of sunlight and

The skin was thick, dry and scurfy, the nose dry and crusty with mucus discharge and the eyes watered

the skin was well lubricated with caron oil.

It was noticed that the blood was very thin and watery. Several smears were examined for *Anaplasma marginale*, but were negative.

In about two weeks, all the affected cows and the bull developed severe cases of scours. Various sulfa drugs, catechu, Krameria, olive oil and nicotinic acid, large doses of vitamin B, and cod liver oil were employed in an effort to check the scours. But none did any good. The fecal material had a most offensive odor.

In the meantime several veterinarians had been called in consultation and the condition was diagnosed as about everything from a run down condition to Johne's disease.

Dr. Frank P. Mathews had just set up his laboratory in Amarillo to study the disease commonly called "wheat poisoning." He was contacted when the outbreak was about over and his council proved very helpful. Doctor Mathews says "Most cases of this ailment start with a hemoglobinuria and a high temperature, but both conditions last only a few hours and are usually not detected." The temperature of all these animals was normal or slightly below normal at the time I was called, which probably in no case was sooner than several hours after the illness began. Five cows, the bull, and six calves died over a period of two months. All ate well until they

dropped dead either in or beside the feed trough. The accompanying illustration shows the last cow that was affected, just before she was sacrificed for necropsy by Doctor Mathews.

His report of the necropsy is as follows:

"The kidneys show a marked degeneration of the epithelium, cirrhosis and a round cell infiltration of the supporting tissue, in other words a parenchymatous and interstitial nephritis which I consider to be typical of our X disease of cattle.

"The liver lesions such as they are, are what I would expect with this disease at the stage in which we killed the animal.

"There is a marked hemosiderosis of the spleen indicating an extensive blood destruction.

"I am inclined to believe that at one time this animal had a hemoglobinuria of short duration during the onset of the disease and that it went undetected.

"This is the disease in which I sometimes find leptospira but their significance has never been determined.

"I am inclined to think that this is a virus disease previously undescribed and that the leptospira are secondary invaders."

By December 15th, the condition appeared to be entirely cleared up and the surviving members of the herd were doing well. However, another case developed January 10th and at this writing (Jan. 15) the owner and I are expecting the worst.

G. B. ADAMS

Vernon, Texas

Double Inguinal Hernia in a Bitch

This case seems worth reporting since the writer has not encountered a similar case nor seen a report of one like it in the literature.

The history of the 12-year-old, female fox terrier brought in for treatment November 4, 1945, was that she had been injured by a car a year previously. The owner reported the dog was ruptured at the time, but until recently no ill effects had been noted. For the preceding two days the bitch had refused to eat and had groaned continuously and for the past three weeks had lain on her back considerably.

Examination revealed a double inguinal hernia. The soft fluctuating mass in the right inguinal region was the size of a man's fist. the enlargement in the left inguinal region was of the size of a hen's egg. Manual manipulations failed to reduce the hernias and immediate operation was decided upon. A prognosis, guarded as to the outcome, was given.

One-half grain of morphine was administered followed in one-half hour by nembutal to effect. On incision over the swelling on the right side, it was revealed that it contained a part of the body of the uterus and about three inches of each cornu. A decomposed fetus was found in the body of the uterus and was removed via the vagina. The uterus was replaced in the abdominal cavity and the inguinal ring was closed with No. 0, 20-day chromic catgut. The skin incision was closed with braided silk, using mattress sutures. The same procedure was followed in the left flank in which the herniated mass was omental fat. The left inguinal ring was closed after replacing the fat into the abdominal cavity.

After care consisted of dressing the incisions with sulfanilamide powder and administration of sulfathiazole by mouth. One tablespoonful of mineral oil was given daily to prevent constipation from inactivity.

The patient was able to move around, reluctantly, five days after the operation and was discharged on November 10th. Complete recovery followed.

J. W. McCOY

Paris, Texas

Relief of Choke by Manipulation

The article on the treatment of choke in cattle in the February issue of VETERINARY MEDICINE recalls various instances in my own practice in this fruit and potato-growing district. I think I have probably had more than my share of choke in the horse and the cow.

In this practice it seems to me in 95% of the cattle that were choked, the obstruction was located in the cervical esophagus where it could be palpated—some "high," some "low." My procedure was to make a cone or point of my fingers and push the apple or potato toward the pharynx. Many times I have pushed in hard with my right hand and if the obstruction were an apple or large potato, my left thumb, pushed in behind it hard, would move it forward. If it were a small or oblong smooth potato, my entire hand with the fingers pulled to a point, but held stiff, would start it moving.

In choke in cows, I worked mostly on the left side of the animal with my right arm over the neck and my right hand pushed hard into the jugular furrow on the right below but against the obstruction. My left hand I kept pushed in and upward and the thing would move about an inch or a little more at each push—but it moved! Usually I don't use a speculum or anything else in a cow's mouth. When the offending body reaches the glottis, the cow first tries to swallow it, and one beads with sweat holding the gain; then she coughs and out it comes.

If and when my hands get so tired I can't move, I try to hold what I have gained until rested. Of late years I have had a block that worked fairly well. A piece of wood, about 2x3x7 inches was rounded on one side and end to fit into the espohageal groove. I push it behind the obstruction and have the owner or attendant hold it steady with one hand in the opposite jugular groove while I flex my fingers and rest them. But it is difficult for many knotty fingered farmers to be of much help, occasionally I find a farmer's son or a city farmer who can hold it as well as I. One summer night the only help I had was a school marm. I tied the nose holder rope to a joist so the cow's neck was held well extended and her head held low. I braced my feet in the concrete feed trough and didn't pause until that apple was up to that cow's pharynx, but she wouldn't cough. She fought and the apple slipped back six inches or more. I then put a speculum on her and opened her mouth wide. I gave my lady assistant a lesson or two and went at it again. When I said "now," Miss School Teacher plunged her arm in that cow's mouth and hauled out the apple and got a nice long red scratch on her arm.

"Pushing them down" never worked satisfactorily with me. I have yet to fail getting an obstruction up even if just palpable at the entrance to the thorax.

Gas pressure from a bloat often helps. It takes strong flexible hands and lots of patience and great perseverance to relieve a choke by manipulation. Just those three things!

P. B. SILVESTER

Princeton, N. J.

A Method of Treating Dislocated Hip Joints

A male hound, two years of age, was presented at the hospital with the following history; about a month previously he had been struck by an automobile and had been lame ever since; carrying the right hind leg practically all of the time.

Examination showed a luxation of the femur with the head above and to the side of the acetabulum. Healing had taken place and the femur had strong attachments in its new position. The leg was shorter than its mate and limited in movement.

After giving an unfavorable prognosis the dog was put under general anesthesia and attempts were made to bring the head of the femur back into place. Traction was unavailing and manipulations of all sorts proved futile. The difficulty was overcome in the following manner: the right leg being dislocated the hound was laid on the table on the left side. With the right hand the right leg was pointed directly at the ceiling, the left arm was now brought around the thigh and the hand grasped the left thigh. Using the left

Fig. 1

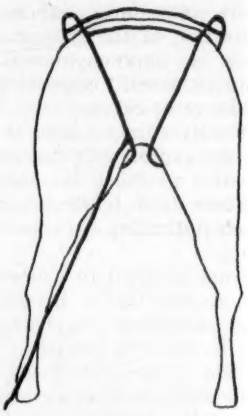


Fig. 2

hand as a fulcrum, pressure was applied with the arm. Grinding and tearing noises could be heard and shortly thereafter the femur popped back into position. Injections of muscotone were made all around the joint, the intent being to produce a swelling that would retain the joint in place.

We pause in the treatment to discuss the difficulties encountered in setting a hip joint. The problem being mainly a mechanical one will be spoken of as such. It has been noted that various means have been employed not only to replace the displaced bone, but to maintain it in position, that normal healing may take place. Several methods have been used extensively the majority of them comprising immobilization of the hindquarters. Mechanically the hip joint is the hinge of the whole leg. Therefore our problem consists of immobilizing the leg and thereby locking the hinge or injured joint in such manner that healing can take place. It was found on examining several cases of dislocated hips that if the leg could be maintained in at least a parallel or even more accentuated position with the opposite leg, and somewhat forward that a week would prove sufficient time to restore the functions of the joint. The dog will favor the leg for at least another week so that gives us two weeks for healing purposes; one week without the encumbrance of a splint, which allows for a slight amount of exercise and "loosening" up of the joint.

The splint shown in the illustrations is made of one piece of wire, heavy enough to fix the

leg in position when applied, yet at the same time flexible enough to be bent to the desired shape. Figure 1 is the splint complete, spread on a flat plane. The dislocated leg is of course fixed in the Thomas splint portion, the circular part on the well leg, and the two bars across the croup forming the means of fixation. Figure 2 shows the splint applied. It will be noted that the leg is fixed by the splint so that the ball cannot slip out of the socket of the hip joint.

In summary it might be mentioned (1) that the splint provides a definite means of fixing the hip joint so that healing may take place, and (2) it provides not only fixation but allows mobility of the hindquarters.

Attempts are being made to devise a splint in three parts, a Thomas splint, two bars, and the circular part of the splint to be connected and adjusted by means of screws. The use of bars across the perineal area and under the belly are also under consideration.

CHARLES L. CRUSE

Winston-Salem, N. C.

Treatment of the Pneumonia-Enteritis Complex in Swine

Recently I have been reading much about a so-called pneumonia-enteritis complex in swine, and treatment for this complex. In no instance have the symptoms been given. I assume that this name is the old timers' "mixed infection" or "barnyard disease."

Regardless of the name used, I feel that these conditions are all of nutritional origin with pneumonia and enteritis as sequels. However, in treatment, the sequels must be treated along with the nutritional deficiencies. One treated without the other will not improve the condition of the animal.

August 28, 1945, in the evening, a farmer stopped in my office and presented the following problem: he had 50 head of spring pigs that had been sick for more than a month. The mortality had been low, but the majority of the pigs weighed only 40 to 50 pounds, although they should have weighed over 100 pounds, as a few did. These pigs had been examined by another veterinarian and a diagnosis of mixed infection had been given. According to the farmer the treatment had consisted of two injections of a mixed bacterin, and a stock tonic. No improvement resulted. Then the hogs were given a therapeutic injection of hog cholera antiserum and continued on the veterinarian's tonic formula, with no benefit to date. Two more pigs had died that day.

The next morning, I examined the pigs. Their general appearance was that of undernourishment and malnutrition. Their eyes were fairly bright, their tails curled, and there was no elevation of the temperature; but they were scrawny, and every so often an occasional cough could be heard. Their feces varied from diarrhea in some to constipation in others. Their appetites were poor. A postmortem examination of three pigs revealed various necrotic areas in the lungs with some adhesions to the pleura. There were no other changes except an occasional slightly inflamed area in the small intestine of one. Some round worms were found in one pig. A live pig was then shipped to the diagnostic laboratory of Pitman-Moore Company.

To arrive at a specific diagnosis was rather difficult. To say mixed infection would have sufficed, but I thought it closer to the so-called pneumonia-enteritis complex in view of the cough, necrotic lungs and enteritis.

While I awaited the laboratory report I put the herd on a strychine-iron-arsenic tonic to try and get them to eating better, and advised adding tankage to increase the protein in the diet.

A few days later the laboratory report arrived. In general this is what it contained. General appearance of malnutrition. Necropsy disclosed necrotic lesions on lungs and pleura. Cultures of the lesions showed Streptococcus and Pasteurella; the lesions being due mostly to the Streptococcus. A few round worms were found in the intestines. There were no other lesions.

The report suggested that no further biological therapy would be of value in view of the history of the other veterinarian's treatment. It suggested adding vitamin B complex to the diet and increasing the protein, especially of animal origin. The laboratory also suggested using their new preparation called di-sufalac. Dose to be 1cc per pound, body weight to overcome the Streptococcus lesions.

I ordered the di-sulfalac and in the meantime told the farmer to continue the tankage in the ration. I dispensed 12 pounds of vitamin B complex powder (Jen-Sal); 1 pound daily in the feed of the herd.

A few days later I returned and gave each pig 1cc per pound di-sulfalac intraperitoneally. The pigs were eating slightly better already.

Two months later I treated these pigs for worms, and found most of them weighing well over 100 pounds and in excellent condition. The farmer was extremely pleased. He said that within a few days after the di-sulfalac injection the pigs began to breathe much better and eat better and from then on they gained weight rapidly.

I am certain the B complex and tankage contributed greatly to the improvement of these hogs, but I believe that the di-sulfalac aided in overcoming the Streptococcus infection in the lungs and thus gave the hogs a chance to survive. I think neither the improved ration nor the di-sulfalac alone, would have done much good.

HARVEY C. TABLEMAN

Warren, Illinois.

Intracisternal Injection of Potassium Phosphate in the Dog

The physiologic responses to intraventricular injection of pituitrin and pilocarpine have been experimentally studied by Cushing. Other investigators had demonstrated that certain substances such as dyes, toxins, and nerotropic viruses produced no effect when injected into the blood stream, whereas when injected intracerebrally, intraecally, and intraventricularly, active responses were obtained. The experimental evidences indicate that there is a "blood-brain barrier" and that this barrier is mediated through the capillaries of the central nervous system which are endowed with a selective permeability.

The recent work of Stern on the direct action of potassium phosphate on the vegetative centers by intraventricular or intracisternal injection suggested their use in shock. By the single injection of small amounts of potassium phosphate into the cisterna magna, she and Chvoles were able to bring the animal out of shock even when the shock was due to loss of blood. The resulting elevation of arterial pressure is attributed to the direct stimulation of the sympathetic centers by the potassium and depression of the parasympathetic centers through the decrease in ionization of the calcium by the phosphate ion. Favorable results in clinical studies on the battlefield were also obtained by the cisternal injection of one to two cubic centimeters of the solution.

In 1943 Smolik made a study to determine the action of potassium phosphate under various experimental conditions, especially hemorrhagic shock. The results he obtained are the basis for this report.

Nine dogs were used, seven of which were subjected to varying degrees of hemorrhage from the femoral artery. Intravenous barbital anesthesia was employed in all experiments. In each instance in which hemorrhagic shock was produced, the dog was bled from one femoral artery so that a blood pressure level of 50mm was maintained for at least one hour and then further lowered to a level of 30mm of mercury for 30 to 45 minutes. In all of these cases when the full amount of withdrawn blood was reinjected and no sustained rise in blood pressure was produced, the dog was considered to be in shock and the solution of potassium phosphate was then injected intracisternally.

Mean blood pressure was recorded with the mercury manometer from the other femoral artery and respirations by thoracic cage pneumograph and tambour. The solution consisted of 1/6 molar (isotonic) solution of potassium phosphate (mixture of KH_2PO_4 and K_2HPO_4 brought to pH 7.6) as prepared by Stern. It was injected by cisternal needle with the dog's head in a lateral dependent position. Stern recommended 1 to 2cc as the therapeutic dose for a man weighing 70 to 75kg. Calculated on this basis, 0.4 to 0.5cc was considered therapeutic for a dog weighing 15 to 18kg. The amount of blood withdrawn varied from 10 to 60% of the estimated blood volume.

Only when reinjection of all withdrawn blood failed to produce an effect was the dog considered in shock. Then potassium phosphate was injected intracisternally. Whenever the pressure was above 50mm favorable responses were obtained. However, when the animal was in a precarious condition, injection of the phosphate was not only ineffective but apparently dangerous. Large doses (approximately four times the calculated therapeutic dose) resulted in dramatic rise in blood pressure but were followed within a short time by death due to failure of blood pressure and respiration. With potassium phosphate Smolik obtained tremendous rises in blood pressure in normal dogs with acceleration of pulse and respiratory rates. Animals in shock, however, did not respond uniformly. At times there were no increases in pulse rate and blood pressure, and in some instances the action was apparently depressant. The conclusion that may be drawn from this experiment is that the direct central action of potassium phosphate is remarkable, although its exact mode of action is not definitely known.

Addendum

Downman and Mackenzie (*Lancet* 245:471, 1943) report the effects of intracisternal potassium phosphate injection in rabbits. These investigators obtained rise in blood pressure, augmented respiration, and increased muscular tone following the injection but they found that these responses were not altered by previous hemorrhage. They found no permanent toxic effects following the injection.

H. J. MOSTYN

Sussex, N. J.

Abstracts

Treatment of Trypanosomiases

Fifty-one blood smears of test animals were examined[21] and 24 were positive for *Trypanosoma congolense*. Tartar emetic was the first drug to prove of practical value in the treatment of animal trypanosomiases and it has contributed to the recovery of countless thousands of fly-struck animals. However antimosan (sodium antimony III biscatechol-2, 4-disulfonate. This is the same as fuadin except that fuadin is a potassium instead of a sodium salt.) given subcutaneously is more easily administered and is much less poisonous than the corresponding dose of tartar emetic, and the trypanocidal action *in vivo* is the same.

* * * *

Vitamin A Deficiency and Acetonemia

An article[27] dealing with research and clinical observation by veterinarians at the New York State Veterinary College is most timely. The report details the symptoms, course, the level of glucose in the blood and of acetone bodies in the blood and urine of ten cases of acetonemia or ketosis which were given enormous doses of vitamin A with little if any beneficial effect. This discussion is a complement to a series of articles by Dr. John W. Patton published in this magazine in 1944 and 1945 in which he reported:

1. There is a broad belt in the United States stretching from the Carolinas to central Oklahoma and from the southern boundary of Kentucky to within a hundred miles of the Gulf in which the farm dairy cows (but not cows in commercial dairy herds) suffer extensively from vitamin A deficiency during the late winter months. In some years vitamin A deficiency is more prevalent and occasions a greater loss in that section than all ailments of dairy cows combined.

2. Vitamin A deficiency as it occurs in the cows in this section is indistinguishable clinically, or by means of the Ross test, from acetonemia or ketosis and is commonly called acetonemia by veterinary practitioners.

3. Uncomplicated cases of vitamin A deficiency make spectacular recoveries when given large doses of vitamin A.

Patton made no report on vitamin A therapy in cases of acetonemia in which vitamin A deficiency was excluded by the adequacy of the ration in this respect.

The present report from the New York veterinarians deals with this phase of the subject. It shows conclusively that acetonemia or ketosis not due to vitamin A deficiency is not significantly benefited and probably not benefited at all by the administration of vitamin A.

Ten cases of acetonemia are reported by the authors. Four of them were complicated or too mild to furnish definite information on the single question of the value of vitamin A therapy. The other six cases, 4, 6, 7, 8, 9 and 10 were uncomplicated cases of ketosis of moderate severity and definitely were not cured and probably in no way improved by the administration of extraordinarily large doses of vitamin A.

The history of the cases is less complete than that designated by Patton as essential to a diagnosis of vitamin A deficiency, but by his criteria would exclude it.

Of case number 4 it is said the cow was "fed much better alfalfa, etc." This would exclude vitamin A deficiency.

Of case 5 the feeding is reported to be "fair in quality and amount." This too would exclude vitamin A deficiency according to the criteria given in the former discussions.

Case 6 was "fed an excellent quality of roughage and the owner was a liberal grain feeder." Again no chance for vitamin A deficiency.

Case 7. "This owner fed excellent alfalfa hay and a liberal grain ration." As in the others the ailment could not have derived from a lack of vitamin A.

Case 8. The "feed was fair in amount and quality." More definite information as to the ration in this case is desirable, but it lacks much of meeting the stipulation of Patton of a high or fairly high producing cow maintained on a diet inadequate in quality or quantity or both over a long period.

Case 9. The owner of this cow "was a poor feeder." This would not exclude vitamin A deficiency, but the diagnosis was made solely upon the finding in the Ross test which Patton found of little diagnostic value in vitamin A deficiency. The eye symptoms, which he says are of the greatest diagnostic value were not present.

Case 10. "This animal was fed hay, silage and plenty of grain." A far cry from leached peanut vines and cottonseed meal on which cows in the acetonemia belt were fed. "Pasture was not too good during August, the month in which this case occurred." This case like the preceding cases does not approach the specification of an inadequate ration "not for

[21] Hourney, H. E. Reflections on the value of tartar emetic for the treatment of trypanosomiases. *Vet. Rec.*, 57:60, pp. 585-586. 1945.—*A. H. Bryan.*

[27] Hayden, C. E., Fincher, M. G., Roberts, S. J., Gibbons, W. J., and Danks, A. G. The value of vitamin A therapy in cases diagnosed as ketosis in dairy cows. *Cor. Vet.*, 36:1, pp. 71-84. 1945.

weeks but for months" given as a requirement for the development of vitamin A deficiency.

This discussion does not deal with the value of vitamin A therapy in the type of cases discussed by Patton, but it does show vitamin A therapy to be ineffective in ketosis not of vitamin A deficiency origin. It supports the stress placed by Patton upon the necessity for an accurate diagnosis and his picturesque but brusk statement that "Vitamin A in the medicine case is not a substitute for gray matter in the cranium."

Corynebacterium Pyogenes Infections of Domestic Animals

Corynebacterium pyogenes[20] is a common cause of suppuration in cattle, sheep, pigs and goats, and is the cause of one form of bovine mastitis, abscess formation, a few cases of abortion, arthritis and granulomatous lesions in cattle; *C. pyogenes* is also associated with calf pneumonia. It occurs in udder and lung infections of sheep, in abscesses and pneumonia in swine and in a mastitis of goats. *C. pyogenes* is one of the commonest causes of pus in cattle whether in the female genital tract or elsewhere.

Animal Infections of Streptococcic Origin and Possibility of Their Transmission to Man

Strangles of horses *(Str. equi)* and mastitis of cows *(Str. agalactiae)* are the most important streptococcic affections in domestic animals.[12] Lancefield after careful study of serology and biological characters of these streptococci divides them into three groups: human streptococci belong to the group A, *Str. agalactiae* to group B, and *Str. equi* to group C. In spite of this classification, there is no doubt that streptococci of horses may infect men, and this happens sometimes to veterinarians. *Str. agalactiae* is regarded as the responsible factor of some human epidemics caused by infected milk. Epidemics of this kind, sometimes serious, have been observed in the United States and Scandinavia. The chief symptom is an inflammation of the throat and adnexa, which may be followed by various complications. Bacteriological examination showed the presence of streptococci not identical to *Str. agalactiae* but with the characters of the hemolitic Streptococcus of scarlet-fever. Therefore, one is induced to think that such epidemics are due to cows infected with human types of the Streptococcus. Possibly the cow plays the part of the intermediary, its infection being due to a human carrier. In order to avoid such epidemics, all persons suffering from sore throat, amygdalitis, or severe colds, and all carriers of pathogenic streptococci should be prohibited from handling dairy cattle or milk.

[20] Lovell, R. *Corynebacterium pyogenes* infections of domestic animals. *Vet. Rec.*, 57:52, pp. 673-685. 1945.—*A. H. Bryan.*

[12] Lerche, M. Les affections vétérinaires d'origine stréptococcique ét leur transmissibilite a l'espece humaine. Deut. *Med. Wochsch.*, Abstr. in *Ann. de Méd.* Vétér., 11, p. 491. 1939.

Spread of Salmonellosis in Chicks and Ducklings

The author[19] reports that a serious outbreak of salmonellosis in young chicks gave rise to a mortality reaching up to 36% due to infection with *Salmonella thompson* and *S. aertryke*. Bacteriological examination of cloacal swabs showed that fecal contamination was a probable source of this infection. Disinfection of eggs within the incubator by formaldehyde gas is suggested as a control measure where the disease is suspected. Fumigation of the chicks during hatching should also be practiced. The spraying of the floor and lower walls of the incubator room, during removal of chicks for packing or sexing may be a useful additional measure. The presence of Salmonella on egg shells may be a method of infection in cases of food poisoning in man.

Avian Goiter

Goiter in poultry has been reported by various investigators as being common in goiterous districts of the United States where goiter is prevalent in other domestic animals. Mortality of birds from goiter has been reported only in exceptional cases and infrequently has lowered production or reproduction been recorded. Only two reports of high mortality in chicks hatched from eggs of goiterous hens have been made.

Since 1929 the authors[26] have noted the occurrence of goiter in pigeons on Long Island and, in recent years, have studied many cases there and in South Carolina. Their experience would seem to indicate that birds are as susceptible to goiter as are mammals, with the inference that there are far more cases in poultry than have been recognized.

The symptoms of goiter in pigeons are, unkempt appearance, lowered production and a high death rate in embryos and in newly hatched birds. The response to the addition of potassium iodide to the ration is the same as in goiterous mammals—prompt recovery.

[19] Wilson, J. E. Infected egg shells as a means of spread of salmonellosis in chicks and ducklings. *Vet. Rec.*, 57:36, pp. 411-413. 1945.—*A. H. Bryan.*

[26] Hollander, W. F., and Riddle, Oscar. Goiter in domestic pigeons. *Poultry Sci.*, 25:1, pp. 19-27. 1946.

Book Reviews

Life and Letters of Dr. William Beaumont. By *Jesse S. Myer,* A.B., M.D., with an introduction by *Sir William Osler,* B.T., M.D., F.R.S. 327 pages. Illustrated. Published by The C. V. Mosby Company, St. Louis, Mo., 1939. Price $5.00.

This is a reissue of the classic work of Doctor Myer originally published in 1912. The reprint contains "A present-day appreciation of Beaumont's experiments on Alexis St. Martin," by the noted physiologist Prof. Andrew C. Ivy of the School of Medicine, Northwestern University, which was not included in the original, and which adds materially to the value of the work.

Of Beaumont, Sir William Osler has said: "The pioneer physiologist of the United States and the first to make a contribution of enduring value, his work remains a model of patient, persevering research." Dr. A. C. Ivy says of him, "One truly does not know gastric physiology and pathology as one should until Beaumont has been read critically and *in toto.*" and again "It is no wonder that a man imbued with such spirit [for discovery of truth] made many observations *all of which have been confirmed*" (ital extra).

The author states that by comparison of Beaumont's work with the most recent developments in the physiology of digestion the scientist "marvels at the thoroughness of this research which was conducted by a young army officer under the most discouraging circumstances."

Yet the work, that earned these encomiums of three competent critics and the acclaim of three generations of the medical profession, was accomplished "under the most discouraging circumstances." Beaumont's laboratory was a trapper's tent or his own living room on a frontier army post. His physical equipment consisted of a "gum elastic tube," a magnifying glass, a scale, a thermometer, a muslin filter, nut gall and two incubators, one a basin of water on a sand bath, the other an arm pit. For a patient he had an undependable, avaricious voyageur whose coöperation was obtainable only for pay, at an ever increasing price, at a time when Beaumont's income as an army surgeon was $200 a year.

The present biography is well and interestingly presented, by a biographer whose penchant for accuracy equals that of his subject and whose industry and discrimination are evident on every page. It will be read as long as physiology is studied and so long as the development of the science of medicine holds interest.

Clinical Laboratory Methods and Diagnosis. A Textbook on Laboratory Procedures with Their Interpretation. By *R. B. H. Gradwohl,* M.D., D.Sc., Director of the Gradwohl Laboratories and Gradwohl School of Laboratory Technique; formerly Director of Laboratories St. Louis County Hospital; Pathologist to Christian Hospital; Director Research Laboratory, St. Louis Metropolitan Police Department; Commander Medical Corps, U.S.N.R. (ret), etc. Third edition, two volumes, 2230 pages, 726 text illustrations, 57 color plates. Published by C. V. Mosby Co., St. Louis. 1943. Price $20.

The author stated in the first edition of this voluminous work that it was "written to help the clinician, the laboratory worker and the medical student to learn laboratory diagnosis. The standard technic of accepted procedures is given in strictest detail, together with an interpretation of all data obtained by standard tests. Necessarily the fields of clinical medicine and surgery have been entered; however no apology is offered for this adventure, since clinical pathology embraces practically every department of medicine."

Any book reviewed in these pages, or any other book if available, may be obtained by remitting the published price to VETERINARY MEDICINE, 7632 S. Crandon Ave., Chicago 49. Bulletins and Circulars should be requested direct from the address given in the notice. Generally they are supplied free unless a price is stated.

This tremendous work ranging far beyond the listing of the technic of the myriad tests of the chemical and biological laboratory. Essentially these ponderous volumes are a group of textbooks on nearly related medical subjects. There are 109 large (6½x10 inches) pages on urinalysis, 221 pages on blood chemistry, 334 on hematology, 118 on blood groups and transfusion; a total of 673 pages on the blood. There are 274 pages devoted to a discussion of bacteriologic applications to clinical diagnosis, 100 pages on serology and almost 400 pages on parasitology and tropical medicine. Among less extended discussions are 20 pages on fecal examination and an equal number on postmortem examinations and on the detection of crime by laboratory methods. There are 15 pages on the preparation of museum specimens and 12 on the use of microscope. The index occupies 100 pages and lists some 18,000 subjects.

Naturally a considerable portion of the work is not applicable to veterinary medicine but much of it is as useful to veterinarians as to physicians. The author claims "today every physician finds it advisable and necessary to conduct routine and laboratory tests." Not all

veterinarians find it "advisable and necessary" to use laboratory tests routinely but indubitably most of them do and to them these volumes will prove no disappointment.

* * * *

Dogs in the News. Compiled by "Dear Dog Lady" (Helene Arlington). 484 pages. Published by the Paebar Company, New York, 1946. Price $3.75.

This work is arranged in two unlike parts. Part I is unique. It is a reprint of items and stories about dogs from newspapers and other publications from each state, the District of Columbia and Alaska. The newspaper clippings include stories of adventure, rescue, heroism, sacrifice, intelligence and loyalty exhibited by dogs, with a sprinkling of strange happenings and some philosophy. They are informative and interesting, some are gems of literature.

But many clippings are reproduced from publications other than newspapers. Many of these are not news but mush; sometimes so deep it gets into the reader's hair. This material is freighted with the blah of talking dogs and letter writing dogs, on the IQ plane of the four-year-old.

Part II contains much useful reference material, the organization of the American Humane Association, brief cynology, some canine history and a discussion of uses of dogs, a list of dog cemeteries, the organization of the American Kennel Club and the names of constituent clubs, major awards at the Westminster show, directions for showing dogs and show philosophy, list of recent dog books, list dog magazines, list of veterinary colleges, list of veterinary magazines, a discussion of dogs and the fancy in Canada and in England, a discussion of obedience training and a highly colored account of War Dogs.

In the long catalogue of the valuable services dogs have rendered man in peace and war, a very important one is omitted—that of serving objectively as man's proxy in the pursuit of scientific knowledge of the functions of his organs and remedies for his ills (and ailments of the dog also). In short the great part that the dog has played in scientific experimentation by which knowledge was gained that has made man's days more comfortable, happier and more numerous is not emblazoned. By this measure, less than justice is done to the dog.

Regrettably a diatribe labeled "antivivisection" is included in which silliness and falsity compete for space and leave little for anything else. But for this bad mannered display of vindictiveness and emotionalism, the work could be recommended for the veterinarian's waiting room.

Animal Breeding Plans. By *Jay L. Lush*, Professor in Animal Breeding, Iowa State College. Third edition. 443 pages, 50 charts and graphs. Published by the Collegiate Press, Inc., Ames, Ia., 1945. Price $3.50.

From the title the prospective reader might expect a discussion of proved bulls and artificial insemination rings, of pasture breeding and flushing ewes. He would be far off the mark. The author tells in the preface that the work has grown out of sixteen years of teaching animal breeding to college students who have already had courses in genetics, embryology, anatomy and physiolology of farm animals, herdbook study, history of the breeds and stock judging.

Much of the discussion is on an erudite plane —a sort of applied genetics. Breeding plans based upon selection, relationship and somatic likeness are considered together with the history and general principles of animal breeding.

The work contains a vast amount of authentic information concerning inheritance in breeds and families, the usefulness and futility of fairs and stock shows, limitations in stock judging, advantages and disadvantages of inbreeding and many related subjects.

The discussion of the rate of improvement which can be expected from various breeding plans and the definite statement of what each method will and will not do in modifying the genetic make-up of the herd should be most helpful to the scientific breeder.

* * * *

Useful Bulletins, Circulars,

To Succeed with Sheep.—Cir. 366, College of Agr., University of Wisc., Madison.

The National Turkey Improvement Plan.—Miscellaneous Publication No. 555. U. S. Dept. Agr., Washington 25, D. C.

Feeding Horses.—Suggested feed combinations under varying conditions to make horses more efficient. Farmers' Bul. No. 1030, U. S. Dept. Agr., Washington 25, D. C.

Report of the Secretary of Agriculture.—Order from the Superintendent of Documents, Government Printing Office, Washington 25, D. C. Price 30 cents.

Report of the Chief of Bureau of Animal Industry, Agricultural Research Administration, 1945.—Order from the Superintendent of Documents, Government Printing Office, Washington 25, D. C. Price 15 cents.

Service and Regulatory Announcements No. 131.—Rules and regulations of the Secretary of Agriculture governing the inspection and certification of dressed poultry, dressed domestic rabbits and edible products thereof and condition and wholesomeness. U. S. Dept. Agr., Washington 25, D. C.

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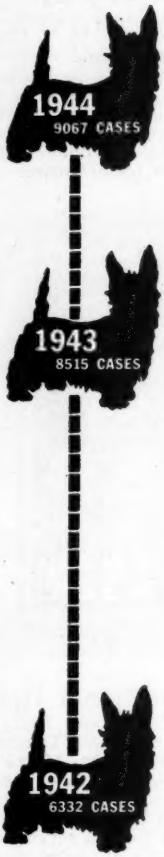

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Immunization of dogs with rabies vaccine is now accepted as an essential measure in rabies control.

RABIES VACCINE *Lederle* has a minimum standard of protection against 10,000 intracerebral fatal doses, or ten times the minimum standard of the U. S. Bureau of Animal Industry.

***Lederle* Laboratories was *first* to develop and distribute CANINE RABIES VACCINE in the United States. Improved methods of manufacture and testing have produced consistently superior vaccines.**

***Lederle* Laboratories was *first* to use a mouse test as a means of evaluating the antigenicity of each lot of rabies vaccine. Subsequently, the U. S. Bureau of Animal Industry required all producers to comply with an antigenic valuation test (the Habel Mouse Protection Test) for rabies vaccine.**

PACKAGES
RABIES VACCINE
PHENOLIZED SUSPENSION 20%

1 dose (5 cc. vial)
6 doses (6-5 cc. vials)
10 doses (50 cc. vial)

Vaccination tag and certificate supplied with all rabbies vaccine.

Listen to the latest developments in research and clinical medicine discussed by eminent members of the medical profession in the Lederle radio series, "The Doctors Talk It Over," broadcast coast-to-coast over the American Broadcasting Company network every Tuesday evening.

LEDERLE LABORATORIES, INC.
A Unit of American Cyanamid Company
30 ROCKEFELLER PLAZA, NEW YORK 20, N. Y.

You'll Be Interested to Know That . . .

4-H Clubs and Livestock Loss Prevention

The National Livestock Loss Prevention Board in coöperation with the extension services of state colleges of agriculture is holding a series of "livestock conservation days" at which awards are made to top ranking 4-H Club teams for achievement in livestock loss prevention.

Speakers on the program include Dr. L. D. Frederick, Chief Veterinarian of Swift & Co.; Dr. W. T. Spencer, Regional Manager, National Livestock Loss Prevention Board and representatives of the traffic department of railroads and of the state college extension services.

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Not So Bad as It Seemed

An article by Dr. Leon Whitney, Orange, Conn., on the care and handling of dogs and other pets appeared in the May issue of the *American Magazine*. The article was elaborately illustrated and recounts experiences of the author as a farmer, dog breeder, geneticist and veterinarian. He does not hesitate to hit hard some of the superstitions of pet owners with regard to their pets. Some unusual experiences are related. A sample:

I arrived at my office one morning to find two elderly women patiently sitting in my waiting room. One of them held on her lap a cage containing a brilliantly plumed parrot. The other, I observed, had a scar on her cheek.

" 'Doctor,' said the first, 'I have a very serious problem. My parrot's upper beak has grown so long it can't pick up birdseed. We have to feed it by hand, one seed at a time, and that takes hours.'

"I suggested that a little careful manicuring might solve the problem, and took the parrot in my hands.

" 'Do be careful,' she warned. 'That parrot is 65 years old. It has been in my family for 40 years and I am pledged to take care of it.'

" 'And be careful it doesn't bite you,' she went on. 'Look at the scar on Mary's cheek. That's what that parrot did to her. It's been an awful trial. I can't have my friends in for fear the parrot will swear. And it swears horribly. It did when I got it. I can't seem to break it of the habit. I don't know what to do, Doctor. I'm simply devoting my life to that parrot and I wouldn't have anything happen to it for the world.'

"Well, something did happen, and in very short order. In the operating-room I delivered the parrot to my assistant, who held it gently. I was about to cut the beak when the parrot gave me a baleful look, uttered a squawk, and died. A bit stunned, I walked out to the waiting-room. 'Madam,' I said, 'your parrot has just died.'

" 'Died,' the woman whispered. 'Died!'

" 'Died,' I repeatedly bravely.

"Suddenly her doleful face lighted up. She clapped her hands and jumped up and down. 'Oh, goody, goody, goody!' she cried."

Veterinary Officer Decorated

Capt. Harry J. Magrane, II, was twice decorated by the Italian government for inspection service in the Italian food depots in Naples and Rome. Captain Magrane has received his discharge and is back in practice with his brother and father at Mishawaka.

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To Make Fur Shows a Safe Place to Display

Fromm Laboratories have offered through the veterinary director, Dr. T. T. Chaddock, to furnish Fox Distemperoid Vaccine and Mink Distemper Serum free to immunize all show animals during 1946. The only requirement is that the material be administered by a graduate licensed veterinarian, selected by official of fur associations, under the direct supervision of Fromm Laboratories.

Veterinary Practitioner Honored

At a meeting of the Mouse River Cattlemen's Association at the State Teacher's College in Minot, North Dakota, March 30, Dr. R. E. Shigley of that city was elected to life membership in the Association "In recognition of upright citizenship and outstanding qualities and in gratitude for his services in good weather and bad which are vital to the welfare of our industry."

Doctor Shigley was born in a log house built by his grandfather at Hart, Michigan. He has practiced in Kenmare and Minot since the year following his graduation from the Chicago Veterinary College in 1909. Since 1916 he has been a member of the state veterinary examiners. He is the eldest of three brothers who are veterinarians—Dr. F. M. Shigley, Inspector in Charge of the federal Bureau of Animal Industry in North Dakota and J. F. Shigley, Professor of Veterinary Science at State College in Pennsylvania, being the other two.